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VOL. I.—16TH YEAR. SYDNEY, SATURDAY, APRIL 6, 1929. No. 14.


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# PEAPES

***— for men AND their sons***

**GEORGE STREET**
**OPP. HUNTER STREET**
**SYDNEY**

# THE MEDICAL JOURNAL OF AUSTRALIA

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# Three Post-Graduate Lectures.[1]

By H. B. Devine, M.S.,
*Honorary Surgeon to In-patients, St. Vincent's Hospital, Melbourne; Stewart Lecturer in Surgery, University of Melbourne.*

## LECTURE I.

### Abdominal Diagnosis from a Surgical Point of View.

#### Introduction.

I must thank you for the compliment you have paid me in asking me to deliver these lectures. I am also glad that you have chosen abdominal diagnosis as the subject, because it is so extremely important and also so pertinent to the everyday practice of our daily lives. You have, however, set me a very difficult task in asking me to cover so wide a field. I propose, therefore, to restrict my remarks to certain abdominal regions and to those difficult problems in abdominal diagnosis which constantly occur in the practice of a busy surgeon, that is to certain phases of surgical abdominal diagnosis which are misleading or difficult to elucidate.

#### General Consideration in Abdominal Diagnosis.

What are the principles that guide us in abdominal diagnosis? They are not the same today as they were twenty years ago. We have had to modify our ideas with the extraordinary progress of surgery. Twenty years ago the surgeon was in the main an operator. Now, he must bring a mind equipped with physiological, pathological and bio-chemical training to bear on his everyday pre-operative, operative and post-operative experiences. He must be an artist in living pathology. The consequence is that the progress of surgery has been more along physiological, pathological lines; along the lines of accurate diagnosis and of better knowledge of the normal functions. And this type of surgeon has done a great deal to bring abdominal diagnosis to a very high pitch. Indeed, the modern abdominal surgeon is very little good to his patient unless he can attack his abdominal problems with a research mind educated and trained in the basic sciences of medicine and surgery. There is a mental clarity inseparable from this kind of scientific training which makes for a physiological dissection of the patient's syndrome, which is, perhaps, the most essential factor in modern abdominal diagnosis. It is not true that the modern surgeon has lost the acute reasoning art of his forefathers. But it is a fact that he is inclined to be obsessed by a host of fanciful diagnostic methods and it is also a fact that he does not always assess these at their relative importance or use them in the right way or to the fullest potentiality, for he fails to use them enough to construct as it were a major syndrome. Indeed the shrewd reasoning that our senior master clinicians developed almost solely by literary study and culture ought to be applied to this.

The history of the patient intelligently taken, symptoms and signs assessed at their proper relative value and physiologically interpreted, scientific examinations, such as X ray, cholecystography, test meal, cystoscopic examination, sigmoidoscopic examination, scrupulously made to "mesh," as it were, with the clinical history so as to make this major syndrome; these are the methods and these are the factors in diagnosis which, after many years of continuous hospital experience, I have come to trust.

A true and pertinent history is a very difficult thing to obtain. The long, complete history is generally stupid and useless and does not develope an experienced mind or experience. Further, a tired historian will not obtain a good history. Time is required, but as a rule, is not taken. Again, it takes an expert cross-examiner to get a good history from some patients. Never let yourself be biased by a knowledge of the case beforehand; this often happens in consultation. Take the history yourself; you may develope quite a different atmosphere because of a fresh mind, unbiased by circumstances. On the other hand, when you have developed your own atmosphere, compare it with and give a lot of consideration to the attendant doctor's point of view; remember he has probably seen the patient for weeks and you have seen him only for the first time. The best way to take a history is not to write it down as the patient tells it to you, but to make a few notes as an aid to memory: to take the history in your mind means that you have to learn the history. When you have elicited everything from the patient and made your examination, write or dictate the history, rearranging it in a coherent way. This is the only history from which you can make proper deductions, and the only history that is of any value when you look at it in three or four years' time, without all the circumstances in front of you. I very often find that, when a history is unsatisfactory, I put it away and take a completely fresh history and I find that some important point in the history is developed which was not elucidated previously.

After the history should come any special examination necessary, such as X ray, test meal, cystoscopic, sigmoidoscopic or other examination. For these there must be some specific indication and the clinical examination and history should always focus these special examinations or they lose a great deal of their value by their diffuseness.

#### A Review Visit.

In a very difficult abdominal diagnosis much time, thought and energy will be expended on the various examinations. It is wise now to allow a little time to elapse; "wisdom comes with night" is a French aphorism and to set aside half an hour for a general review of the whole, let us say, major syndrome. It takes time to think. This will allow time to think, to balance, to coordinate and to assess the value of the findings in the various minor syndromes and to make deductions therefrom. For

[1] Delivered at the Post-Graduate Course of the Queensland Branch of the British Medical Association in August, 1928. The lectures were illustrated by lantern slides.

example, the patient has had attacks of colicky umbilical pain, a cholecystogram has been made and the gall bladder shown to be very low, the alimentary canal has also been examined radiographically and the ileo-caecal junction and appendix visualized and localized in the pelvis. It then becomes a physiological problem to think out whether either of these organs in their abnormal positions can give rise to the clinical picture. Again, the patient has præcordial pain and symptoms like angina. The gall bladder is cholecystographically localized high and to the left side and there are indefinite signs of cholelithiasis. There is perhaps here a high innervation of the gall bladder. Will this explain the angina-like symptoms? It is again a question of physiology. The following case histories will serve to illustrate this.

A young woman had symptoms definitely diagnosed as anginal. We found by cholecystographic examination that her gall bladder was full of stones. She had none of the ordinary symptoms of gall bladder disease, but it was possible to construe the clinical history as being caused by the diseased condition found. She got better when her gall bladder was removed. On the other hand, the mere presence of gall stones is not enough.

A man had anginal symptoms. A cholecystogram revealed his gall bladder packed with stones and he was sent for operation. His anginal complex could not be physiologically construed as due to cholelithiasis and he had cardiac stigmata. I did not operate. He died of heart failure a week later.

A patient had an extremely large gastric filling defect in the præpyloric region. It was typical of carcinoma. The patient had symptoms very characteristic of carcinoma, but the filling defect was in such a position in front of the spine and so large that it should be palpable, yet clinically it could not be felt. Therefore a diagnosis of carcinoma should not have been made, though it was. The clinical and special examination would not as it were mesh. Autopsy revealed Addison's disease.

I give you these examples to show you that it is the brain pregnant with thought that is potent in diagnosis rather than a multitude of incompletely reasoned out and imperfectly coordinated examinations.

### Primary Considerations: Exclusion of Extraabdominal and Parietal Conditions.

It is a basic consideration in abdominal diagnosis to exclude (*a*) general conditions, such as toxic, infective, degenerative and analogous affections, (*b*) the more local diseases of the adjacent regions, such as thoracic affections, (*c*) lesions of the abdominal parietes.

It is in connexion with the last of these that we often err.

The abdominal viscera innervated by the involuntary nervous system give characteristic manifestations in visceral disease, easily distinguished from somatic disease. These are pain, non-segmental, unrelated to movement, indefinitely localized. This is accompanied by nausea, vomiting, shock and sweating, alimentary canal inhibition (at least in some of its segments), secretory disturbance, protective rigidity, deep tender spots. There is also a definite and consistent relation to the intake of food or discharge of air or excreta. Symptoms due to disease of the abdominal parietes are almost exactly the reverse of this. Many grave errors in diagnosis are made by not keeping in mind this primary consideration. I can perhaps best emphasize the importance of this principle by an example in which somatic disease simulated splanchnic disease.

A patient, *ætatis* forty years, who complained of right-sided lower abdominal pain, was sent by a physician to a surgeon to have his appendix removed. Operation disclosed the not unusual equivocally diseased appendix. The right-sided pain recurred. A pyelogram was then made on the ground that as the pain went through to the back, it might be caused by some renal condition. The pyelogram was interpreted as indicating a lesion of the kidney. Apparently, on this evidence, his kidney was removed. The pathologist pronounced it normal. Unfortunately, after two brilliant operations and some dexterous ureteral catheterization, to the disappointment of the surgeon and the patient, the pain returned almost immediately. A review disclosed the facts that this pain was aggravated by movement, was relieved by rest, was segmental in its distribution and was probably somatic in origin. The antero-posterior skiagraphic view contained a clue which had been overlooked, namely, a narrowing of one intervertebral space. This suggested a lateral view which revealed the gross lesion (see Figure I).

A patient, aged thirty-three years, complained of pain over the back in the region of the kidney. He was immediately sent for X ray examination. This revealed an equivocal shadow which was thought to be a stone. At operation no stone was found. If, instead of this precipitate radiography, half an hour had been spent on a study of his pain, quite a different clinical atmosphere would have been developed. The pain had no visceral characteristics whatever and there were the signs of spondylitis, some rigidity in the neck, in the back, exacerbations of pain with spinal movements, periods of relief with rest and a tendency to bilateral symptoms. Here I show you his skiagram in which you can see how gross is the spondylitic change.

Spondylitis is much more common than is generally supposed and in my experience is the explanation of many unsuccessful abdominal operations. Garvin[1] says that in a series of over two thousand patients of over fifty years of age the skiagrams disclosed hypertrophic arthritis in 40% in women and 67% in men. Of the men 74% and of the women 61·5% had no symptoms due to this.

In another patient the root pains of a tumour of the spinal cord at the level of the seventh and eighth dorsal vertebræ were regarded and treated for two months as the dyspepsia of a duodenal ulcer.

### Tumours of Abdominal Wall.

Even tumours of the anterior abdominal wall are not always easy to distinguish. A woman of forty-eight complained that a week previously a pain developed to the left of her umbilicus. There was no history of injury. Her medical attendant found a tumour which he diagnosed as hydatid of the abdominal cavity. She had a hæmatoma of the rectus muscle. Occasionally this occurs in old feeble people with only slight strain, generally after being in bed for some time. In these it is diagnosed as carcinoma of the large bowel because of the age of the patient, the indefinite edge and superficial position of the tumour. The thick rectus sheath generally prevents the extravasated blood from

staining the adjacent tissues. Fixation to the abdominal wall, diffuse edge, absence of respiratory movement, superficial character of the swelling are the characteristics.

I wish now to discuss some of the diagnostic difficulties which occur in certain abdominal regions.

### Œsophageal Lesions with Abdominal Symptoms.

Although the œsophagus is not an abdominal organ, yet it often gives rise to symptoms interpreted as abdominal and it therefore enters into our discussion on abdominal diagnosis. Cardiospasm is often clinically overlooked or misdiagnosed as some abdominal disease.

The history of one patient is most interesting in this relation. She had what she called "indigestion" and had been vomiting continually for some time. As she also had a slight menorrhagia, she visited a gynæcologist. Assuming that her dyspepsia was caused by her uterine condition, the gynæcologist removed a diseased uterus. She continued to vomit in an alarming way for five weeks after her operation. Investigation showed that this was not true vomiting, that the food just regurgitated and did not taste sour. From the skiagram it is evident that she is suffering from very gross cardiospasm or achalasia. Except that nutritional disturbance was more prominent, a patient whom I saw a few years ago, with a somewhat similar condition, had had her uterus suspended because of her so-called dyspepsia. Her symptoms had continued and although she had consulted many doctors, no one had discovered that she had a very extensive cardiospasm.

I intend these cases to show also how easy it is to overlook a cardiospasm as the cause of ill health.

The ætiology of a dysphagia is always a most difficult problem in diagnoses and entails great responsibility.

### Confusion of Malignant Disease of the Œsophagus and Cardiospasm.

The œsophagus is much better supplied with sensory nerves than the stomach. The œsophagus is sensitive throughout its whole length to pressure and temperature, and as the œsophagus, unlike the stomach, can localize a hurt, therefore a patient can generally point to the site of an obstruction in the œsophagus. The diagnosis of the situation of a dysphagia is, therefore, not difficult. Obstruction in the lower end of the œsophagus is especially a difficult problem because of the frequency of cardiospasm in this situation. As a matter of fact a diagnosis may sometimes not be arrived at except by an œsophagoscopic examination. I have seen patients suffering from cardiospasm whose condition had been diagnosed as malignant disease of the œsophagus, "cured" by a herbalist simply because they did not die as their doctor said they would. It is worth remembering that cardiospasm is much more frequently present than is generally supposed. May I narrate to you an interesting case history to illustrate this point?

A man, aged fifty-five years, cachectic and emaciated, had an enormously dilated stomach, and appeared to be in the last stages of pyloric carcinoma. Unexpectedly, the operation disclosed that the pyloric obstruction was caused by scar of healed ulcer, really a reprieve. Gastro-enterostomy was performed under local anæsthesia. Two months later he complained of difficulty in swallowing and in two months complete obstruction of the œsophagus gradually developed.

It was thought that a carcinoma of the œsophagus arising coincidentally with the pyloric scar had been the real cause of his malignant appearance and symptoms. Gastrostomy was carried out. Figure II is the skiagram of his œsophagus.

Notice the curious lower end which should be suggestive of carcinoma. Two years later the patient was so well that his family doctor, thinking the obstruction must have been cardiospasm, allowed the gastrostomy opening to close up. He again became very ill and cachectic, developed mediastinal symptoms and opacities through the lung and appeared to be in a dying condition. Gastrostomy was again performed as a forlorn hope. The patient is still alive, three years from the onset.

Is this a slowly growing scirrhus or is it cardiospasm? The man's condition was always so extremely bad at the time of treatment that it was never possible to make an œsophagoscopic examination. Only the other day was an œsophagoscopic examination possible, enabling a certain diagnosis of cardiospasm to be made.

### *Differential Diagnosis Between Cardiospasm and Carcinoma of the Œsophagus.*

May I contrast for you the differential diagnosis of these two conditions in tabular form?

Table I.

| *Cardiospasm.* | *Malignant Disease of the Œsophagus.* |
|---|---|
| Situated at the hiatus of the diaphragm or at the cardia. | Any other œsophageal situation is not cardiospasm. |
| Bougie may be passed. | Bougie cannot be passed. |
| More frequent in females (neurotic) and young people. | More frequent in males over forty years. |
| Onset sudden; the symptoms remain the same. | Onset gradual and progressive course. |
| Pain substernal, sharp and lancinating and often symptoms of vagal nerve stimulation. | More discomfort than pain. |
| Regurgitated material tastes sweet and smells sweet and is copious. The dilatation is big. | Regurgitated material smells bad and tastes nasty (from ulceration) and is scanty, as dilatation is moderate. |
| Patients look fairly healthy even after twelve or eighteen months. | Patients look very ill, often cachectic after a few months. |
| Wasting not so evident. | Wasting very evident. |
| A glass of water may enable patient to swallow. | A glass of water will make no difference. |
| ? Remissions. | No remissions. |
| May improve after a bougie is passed. | |
| No enlarged glands are seen. Characteristic skiagram. | Enlarged glands in the neck may be seen. Characteristic skiagram. |
| Œsophagoscope discloses no obstruction but greatly dilated œsophagus. | Œsophagoscope encounters obstruction and small piece can be removed and examined under the microscope. |

### Œsophageal Carcinoma Without Dysphagia.

While scirrhous carcinoma of the œsophagus always causes dysphagia early in its course, fungating carcinoma, in which ulceration usually predominates, may manifest obstruction only as a terminal symptom or not at all. I have seen three patients suffering from this kind of malignant disease of the œsophagus whose condition was diagnosed as carcinoma of the stomach and an exploratory operation carried out. In one, although no diagnosis was made at operation, symptoms of dysphagia developed two months later and an œsophagoscopic examination disclosed a fungating carcinoma. In another, also explored, the diagnosis was made only *post mortem*. Profound constitutional disturbance from hæmorrhage, ulceration, sepsis and cancer metabolites of the soft, rapidly growing carcinoma, are responsible for a group of symptoms which simulate those of fungating carcinoma of the body of the stomach.

It should be instructive to contrast for you the X ray appearances of a cardiospasm and of a carcinoma, because X ray examination is such an extremely valuable aid in diagnosis and because it may be necessary to rely on this entirely when it is not possible to get an œsophagoscopic examination. The obstruction occurs at the diaphragmatic opening and the lower end of the œsophagus, approximating the obstruction, not being infiltrated, is bellied out and the barium issues through the obstruction in a fine, even, straight, sharp-edged stream.

In the X ray appearances of carcinomatous strictures it is seen that the narrowing is not exactly at the diaphragmatic opening and that the lower end of the œsophagus, being infiltrated, tapers into the obstruction. The shadow is seen terminating in a cone pointing downwards and from the apex of this a fine twisted stream of barium is seen making its way through the tortuous vagaries of a narrow, irregular, diffuse-edged constriction. The dilatation of the œsophagus is also very much greater in cardiospasm than in carcinoma.

### Gastro-duodenal Syndrome.

There is no sense in considering duodenum apart from stomach. Both are developed from the fore gut. The physiology of both is much the same. Their diseases present the same difficulties in differentiation from other diseases. Grouping them together has the advantage that in diagnosis the more we get down to broad fundamental distinctions, the sounder will be our conclusions.

#### *Dyspepsia.*

Dyspepsia or indigestion may be the first indication of a gastro-duodenal disorder, but it is also caused by a host of troubles. Indeed, the stomach which is necessarily highly sensitized and delicately mechanized for conservative reasons and for the reception of nourishment, is the first organ to be disturbed and the first function to be sacrificed when the body is put on the defensive in the beginning of any diseased state.

Under the term dyspepsia we include a group of symptoms which may indicate any type of gastric disorder, primary or secondary. When the dyspepsia is associated with definite pain, not the fullness or discomfort often described by the patient as pain, I should like to call it painful dyspepsia. When there is just discomfort or fullness and no pain, I call it painless dyspepsia. This, I think, is a distinction which is very practical. The first is always surgical and the second, with the exception of silent gastric carcinoma, generally medical. For example, the syndrome of chronic gastric ulcer gives a painful dyspepsia, while that of functional disease of the stomach, except in the dyspepsia associated with hypertonic stomachs, gives a painless dyspepsia.

#### *Distinction Between "Medical" Dyspepsia and "Surgical" Dyspepsia.*

Toxic or anæmic states may give a painless dyspepsia, which is medical, but not functional, and must also be distinguished from organic gastric disease. The following conditions have at some time or other come under my notice, in patients sent for operation with a diagnosis of gastric ulcer or carcinoma: (*a*) Tuberculous disease of the lung, in the early stages, (*b*) cirrhosis of the liver, (*c*) subacute nephritis with vomiting, hæmatemesis, albuminuria, (*d*) interstitial nephritis with high blood pressure, chronic uræmia symptoms, constant vomiting, loss of appetite, loss of weight, cachexia, (*e*) anæmic states, such as pernicious anæmia.

Two types of functional dyspepsia seem to be constantly appearing in surgical practice. The first generally presents the following symptoms and signs. The patient is generally a woman or, if a man, he is a definite sympathetic type with a low threshold to stimuli of his nervous system and probably has an irritable or highly developed sympathetic system. These patients complain of discomfort after meals rather than pain; they give a history of many years' dyspepsia; their dominant feature is nausea and vomiting. Their symptoms come on immediately after food, that is there is no time interval. Operation reveals a large hypotonic stomach dilated and always full of air. These belong to a type with its own peculiar mental, physical, metabolic and gastric features.

The second type is almost exactly the reverse and has been called sthenic dyspepsia by Hurst. It occurs more often in men or in women with irregular feeding habits. It may give rise to a painful dyspepsia, indistinguishable from that of gastric or duodenal ulcer. The pain, like in ulcer, may come on a definite time interval after taking food. The stomach is small when seen at operation, is very muscular, empties fairly rapidly and very often has a præpyloric blush. This kind of stomach has a highly acid secretion and is often associated with an acid gastritis (described by Aschoff); it is the type of stomach that is very often seen associated with an ulcer. This condition is of interest to the surgeon because, as it gives rise to a painful dyspepsia, it is often confused with gastric and duodenal ulcer. This is the kind of stomach one finds when opera-

tion has failed to disclose the ulcer that has been diagnosed. Some of these highly acid hypertonic painful dyspepsias in my opinion ultimately lead to duodenal ulcer. The pain is probably caused by an incoordination in the neuro-muscular mechanism.

This one form of functional painful dyspepsia, having been eliminated, it may be said speaking on broad lines that all the rest of the painful dyspepsias concern the surgeon.

### Reference.

(1) Garvin: *Archives of Surgery*, July, 1927.

## LECTURE II.

### Abdominal Diagnosis from a Surgical Point of View.

#### Reflex Dyspepsias.

Before the diagnosis of organic disease of the stomach is dealt with, it is necessary to speak of reflex dyspepsia because in quite a majority of cases it is quite impossible or very difficult to distinguish some of the reflex dyspepsias from organic disease of the stomach. The mistake is really not so serious, as the treatment of nearly all reflex dyspepsias is surgical.

I wish to remind you that the alimentary canal is a system of interdependent locks, the food passing from one lock only when the next is ready to receive it. I also wish to refresh your memory of the innervation of the alimentary canal, a complicated, delicate nerve mechanism that controls the coordinated, sequential emptying of these segments.[1] You will remember that the sympathetic part of the autonomic system seems to be concerned with the mechanism of retention and the vagus with the mechanism of emptying. It is thus easy to imagine how disease in one alimentary segment in the same splanchnic field may cause increased retention in another segment which is in the same splanchnic nerve field. You know that in resection of the small intestine or in a case of acute appendicitis a gastric retention of from six to eight hours may occur. Probably this is really a conservative reflex.

Reflex dyspepsia is in most instances painful, first, because there may be incoordination and painful cramp of the neuro-muscular mechanism of the stomach, second, because there may be pain due to inflammation or some lesion in the organ itself. For example, a certain type of cholelithiasis, in which the gall bladder is packed tightly with stones, may give a syndrome of painful dyspepsia exactly like that of gastric ulcer; the pain is here due to a painful functioning in a gall bladder with some gross lesion. In the usual kind of cholelithiasis a mildly painful dyspepsia may occur, probably resulting from a neuro-muscular incoordination and the same is true of appendiceal lesions. Epigastric pains some hours after meals may be due to gross chronic disease of the appendix, whereas a mild painful dyspepsia may be caused by a chronic appendicitis reflexly causing some gastric dystonia.

[1] At this stage the lecturer showed several diagrams.

#### Recognition of Organic Disease of the Stomach.

The only gross organic diseases of the stomach that are not associated with a painful dyspepsia, are acute gastric and duodenal ulcer and some forms of fungating gastric carcinoma of the fundus—silent carcinoma. Perforation or profound hæmatemesis is not unusually the first symptom of acute gastric or duodenal ulcer, perhaps preceded for a few weeks by a very mild painless flatulent sort of dyspepsia.

The edges of an acute ulcer are not infiltrated with inflammatory products; in the base of the ulcer the *muscularis* is plainly seen. Therefore there is no infiltrating pressure on the gastric nerve receptors so sensitive to tension and consequently there is no pain, as there is in chronic ulcer. The multiplicity of acute ulcer indicates that it is often due to a systemic cause or infective focus. In one instance the ulcers actually occurred during the course of an acute appendicitis. Indeed, this kind of ulcer is better termed an infective ulcer. Apart from many other considerations it is necessary to make the diagnosis of acute as against chronic ulcer carefully, because while gastro-enterostomy might justifiably be associated with suture of a perforated chronic ulcer, it would be quite wrong in my opinion to do it with suture of a perforated acute ulcer.

#### Syndrome of Hæmatemesis and Melæna.

There is the greatest difficulty in descriminating between surgical and medical disease of the stomach in connexion with hæmatemesis and melæna. Toxic states either from autointoxication or from ordinary infection cause dyspepsia. Acute gastric or duodenal ulcer, often a consequence of these lowly resistant states, may coexist and since hæmatemesis is a most prominent symptom of an acute ulcer, it is easy to understand why on the symptom complex of dyspepsia and hæmatemesis the surgeon may be requested to operate for a gastric ulcer. It is true it may be an ulcer, but it is acute, non-surgical and infinitely more safely treated by the physician. To mistake the diagnosis and operate for a surgical ulcer is very serious and, moreover, while the operation is physiologically not indicated, it may kill the patient because of the associated disease. Indeed the diagnosis as to whether a hæmatemesis is medical or surgical may be of supreme importance. Most cases of hæmatemeses are, in my opinion, medical problems and the patients die if operated on.

Bleeding from a chronic gastric ulcer is often a most urgent indication for operation. Here I show you the history of a very interesting case. Because this patient had constant attacks of profound hæmatemesis, even though he had no pain with his dyspepsia, he was sent for operation on the assumption that he had a chronic gastric ulcer. The clinical syndrome of this patient might easily have been classed as non-surgical acute ulcer or one of the medical states which give rise to hæmatemesis. It

was found that the spleen, which was not obvious clinically, was seen very distinctly on the radiographic screen. He was suffering from splenic anæmia and needed a splenectomy. The hæmatemeses of (*a*) chronic gastric ulcer, (*b*) gastric carcinoma and (*c*) splenic anæmia are the concern of the surgeon.

*Exclusion of Malignant Diseases.*

The surgeon's attention having been focused on the stomach because of a more or less painful dyspepsia, his essential problem first of all is to establish or exclude a diagnosis of malignant disease of the stomach, for in this disease prompt surgical treatment is imperative and an incorrect diagnosis is almost a fatality. Indeed, so great is the fear of malignant disease that most of the patients who seek medical aid for gastric disorders, demand its exclusion. The early diagnosis of malignant disease, the only diagnosis that is of any value, is extraordinarily difficult and you would be surprised how often gastric carcinoma is undiagnosed.

**Gastric Tumour.**

The absence of a tumour does not help; tumour may be absent because the carcinoma may be on the posterior wall of the stomach, up under the ribs in the vicinity of the cardiac orifice or the carcinoma may be only a flat submucous infiltration or a small scirrhus of the pylorus.

These two histories, illustrating instructive mistakes, are pertinent. The patient had a very indefinite syndrome, a definite secondary anæmia, loss of energy, loss of appetite, a mild painless indigestion, but no tumour. The skiagram revealed a big filling defect. This patient's condition was diagnosed in surgical examination for honours as pernicious anæmia. It is an interesting clinical fact that when her stomach was full, no tumour could be felt; when it was empty a tumour could easily be felt. The growth was on the posterior wall.

Here is the history of a man who was under the care of consultants for twelve months before a diagnosis was made. This man had loss of health and strength, but had practically no dyspepsia; he had no tumour and an antero-posterior skiagram failed to reveal a lesion, but from the lateral skiagram the deformation of the posterior wall can be seen.

*Silent Carcinoma of Stomach.*

Of course both the cases just quoted are examples of that kind of fungating carcinoma of the fundus of the stomach which runs its course almost without producing any localizing symptoms. A profound constitutional disturbance and an anæmia are its chief characteristics. Hydrochloric acid is almost always absent, but this, as you know, does not distinguish it from pernicious anæmia with which it is frequently confused. Ulcerations, bleedings, hæmolysis and metabolic disturbances give rise to these symptoms so suggestive of pernicious anæmia.

*Carcinoma with Symptoms Like Gastric Ulcer.*

When patients with a definite painful dyspepsia present themselves, it is quite a difficult problem to exclude carcinoma of the stomach. My own observations lead me to believe that some malignant growths of the stomach give rise to a painful dyspepsia almost indistinguishable from ulcer. This happens when the carcinoma infiltrates and ulcerates the lesser curve, that is the gastric canal or when it originates in this region as a curious, cartilaginous, flat, submucous infiltration. Probably the distinctive gastric ulcer-like pain is caused by an incomplete interference with peristalsis. It is not uncommon to find these patients medically treated for gastric or duodenal ulcer till time disproves the diagnosis and terminates the treatment.

*The Responsibility of Making an Ulcer Diagnosis.*

Regarded from the point of view of exclusion of malignant disease, the responsibility of making a diagnosis of gastric ulcer and treating it medically, is very great. Here are case histories of instructive diagnostic mistakes which should serve to illustrate this point.

Mr. S.—In May of 1926 a man, aged forty-three years, complained of what he called a cold in his stomach. He could not eat the usual things; they gave him a pain at once and also a pain two or three hours afterwards. He had bad tasting eructations and he vomited occasionally in relation to meals. A test meal yielded the following information: at one hour, free acid 21, total acid 54; at two hours, free acid 40, total acid 58. X ray observations revealed peristaltic waves passing to the pylorus, but they were slightly interfered with by a persistent præpyloric spasm—a rather wide *incisura*. The X ray diagnosis was præpyloric ulcer (see Figure III). Six months later it was noticed that the upper part of his abdomen was somewhat rigid. He had no tender spot. Rigidity in absence of tender spot suggests carcinoma rather than ulcer. This man was under medical treatment for ulcer for fourteen months and died of cancer in March, 1928.

Here is the history of another patient whose affection for twelve months was regarded as a gastric ulcer. His first symptoms were fullness and very definite pain in epigastrium four hours after meals. He began to vomit six months after the onset of his symptoms at first every few days and during the last three months he vomited four to six hours after food every day. The vomiting relieved the pain. There was no occult blood in his fæces. He had lost 12·7 kilograms (two stone) in weight in twelve months; his appetite had been bad for six months and he had been unable to work for twelve months; he had always been constipated, but has had to take a great deal more medicine lately. His blood pressure was 100 millimetres of mercury. There was a general epigastric rigidity, but no palpable tumour and no tender spot. It should be noted that the progressive history, the not very severe food pain or food ease, the epigastric rigidity and the constitutional symptoms, should have suggested carcinoma. This man was treated for twelve months for gastric ulcer. At the end of twelve months it was possible to remove his carcinoma. What a very much better prognosis there would have been twelve months earlier!

Here again is a history of a man, aged thirty-five years, who had been ill for nine months suffering from bad epigastric pain at 11 a.m., 5 p.m. and 10 p.m., relieved by food and an alkaline powder. He had scarcely any loss of weight and a fair appetite. No one concerned with the case had the least doubt that it was anything else but a duodenal ulcer. As he was not getting better after nine

months' treatment, operation was decided on. A flat submucous serviette ring-like carcinomatous infiltration was found at operation.

*Clinical Distinction from Ulcer.*

Doubt should arise in a gastric ulcer diagnosis if: (i) a pain occurs immediately after meals or even if some pain occurs immediately after meals in addition to a pain two or three hours after food, or if there is no clean-cut consistent time interval, (ii) there is much nausea and sickness, (iii) the constitutional symptoms are pronounced, (iv) the whole history is relentlessly progressive, (v) the blood pressure is low, (vi) there is rigidity in the upper part of the abdomen and yet no tender spots, (vii) there are no remissions.

It is unfortunate for the early diagnosis of carcinoma that this peculiar flat submucous infiltration with no tumour is prone to occur in young people. It is, therefore, not surprising that the combination of youth, painful dyspepsia and mild sharp-edged deformation seen in a gastric skiagram often makes for a gastric ulcer diagnosis. The likeness is sometimes very great, because this type of cancer in its early stages is not associated with much constitutional disturbance, as for example in the following case. A man, aged thirty-five years, who came to our clinic because he was not feeling well, was red in the face and healthy looking. He had no detectable tumour even under an anæsthetic. In Figure IV it will be seen that a later and more defined stage of this sharp edged indentation to which I have already alluded, is present. I need not remind you to contrast this with the usual X ray appearance of carcinoma which nearly always gives a diffused-edged deformation. When this kind of carcinoma which is often slow growing, occurs in old people, the initial symptoms which attract attention, are often anomalous and as the skiagram for the reasons given above does not always clarify the diagnosis, some interesting mistakes are made. Here is one.

A patient aged sixty years complained of a boring pain in the mid-sternal region and had a little epigastric tenderness. A few days later he became dazed, lost his memory, appeared strange in manner and started giving away five pound notes. He vomited occasionally and became constipated. Except for a slight nystagmus he had no neurological signs, no optic neuritis and the cerebro-spinal fluid was normal. The condition was diagnosed as encephalitis independently by two consultant physicians. Gradually he began to improve, physically and mentally. Because of his vomiting, he was examined by means of X rays. On two occasions a sharp-edged, spasm-like filling defect was radiologically diagnosed as præpyloric spasm. When I saw him I had no doubt in my mind that his clinical diagnosis was carcinoma of the stomach. I was able to construe the X ray evidence of præpyloric spasm as that sharp-edged deformation due to submucous infiltrating carcinoma. In the light of this diagnosis his supposed encephalitis could be interpreted as a thrombosis of some of the vessels of the frontal area.

Here a cachectic thrombosis of a cerebral vessel ushered in a carcinoma of the stomach exactly comparable with the apparently idiopathic thrombosis of a femoral vein which Sir Berkeley Moynihan says should always make us think of carcinoma of the stomach.

**Gastric Ulcer Giving Symptoms Like Gastric Carcinoma.**

Gastric ulcer in the fundus of the stomach may often give symptoms indistinguishable from those of gastric carcinoma. The ulcer of the posterior wall does not give the sharp, clear-cut, severe pain of ulcer of the lesser curve and when it penetrates the pancreas, the pain is present all the time and made worse by meals just like the pain of gastric carcinoma. To show you how difficult it is to make this distinction clinically and, I might also add, radiologically, I would like you to peruse the following history and mentally to make your diagnosis:

T., *ætatis* thirty-six years, had been ill for years with epigastric pain which came on in bouts, a half to one hour after meals. This pain was relieved by food and magnesia. He had a hæmatemesis years ago, otherwise he had not vomited. Two months ago his illness changed. He began to vomit after meals, the pain shifted to below and to the left of the umbilicus. He began to lose weight and had lost 6·35 kilograms (one stone) in the two months. He now looked cachectic and malignant. A test meal revealed practically no free hydrochloric acid. The skiagram showed only a dilated stomach.

Here there was a large ulcer of the posterior gastric wall penetrating the pancreas. This ulcer was radiologically difficult to see and presented the constitutional and other symptoms of a carcinoma.

**Gastric Ulcer Syndrome.**

I need not touch very much on the symptomatology of chronic gastric ulcer. This is always very clear; the pain is very definite, very consistent, has a definite time relation between the intake of food and the onset of pain, great relief on vomiting; indeed, the patient can almost tell the time of the day from the onset of his pain. Constitutional symptoms are not severe, in fact patients may often increase in weight because they sometimes take food to relieve their pain. Bleeding is not a prominent feature. It is only when the ulcer is high in the fundus, which is very unusual, that the symptoms are indefinite and like those of malignant disease and it is only when the ulcer penetrates and adheres to an adjacent organ, like the liver or the pancreas, that the pain is present all day long and made worse only after food and that the innocent ulcer is difficult to distinguish from malignant ulcers, but this is not of great practical importance because, as a matter of fact, both conditions urgently need operation.

**The Vagaries of Pyloric Stenosis.**

The onset of a malignant pyloric stenosis is often insidious and misleading as, for instance, in this history:

N., *ætatis* thirty years, complained of indigestion, fullness and discomfort from five to thirty minutes after food. This was associated with a mild epigastric pain and much nausea and vomiting, the latter occurring at night some hours after food. Vomiting gave great relief. He was very constipated, going up to four days without a motion.

This patient was treated for many months for dyspepsia and then for a period as gastric ulcer.

## ILLUSTRATIONS TO THE ARTICLE BY MR. H. B. DEVINE.

FIGURE I.
Vertebral Lesion which Simulated Visceral Lesion.
Lateral view of lumbar vertebræ.

FIGURE II.
Lesion at Lower End of Œsophagus.

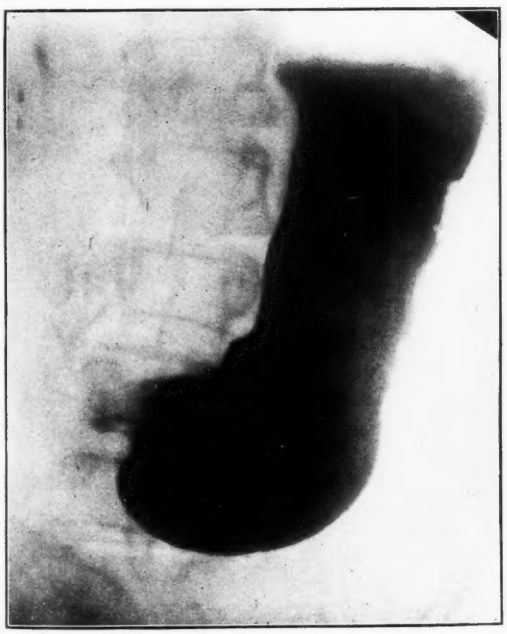

FIGURE III.
An infiltrating type of pyloric carcinoma which may occur in young men and simulate ulcer.

FIGURE IV.
A later stage of the same type of carcinoma as in Figure III occurring in a young man.

## ILLUSTRATIONS TO THE ARTICLE BY MR. H. B. DEVINE.

FIGURE V.
Skiagram Illustrating the Rugæ in the Stomach.

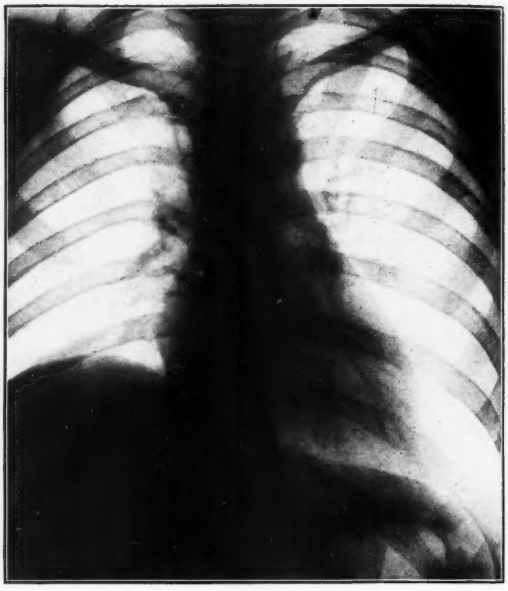

FIGURE VII.
Skiagram showing the raised upper surface of the right lobe of the liver from a central hydatid.

FIGURE VI.
An apparently normal cap in which an ulcer was found.

## ILLUSTRATIONS TO THE ARTICLE BY MR. H. B. DEVINE.

FIGURE VIII.
Early Carcinoma of Sigmoid, Indicated by Arrows.

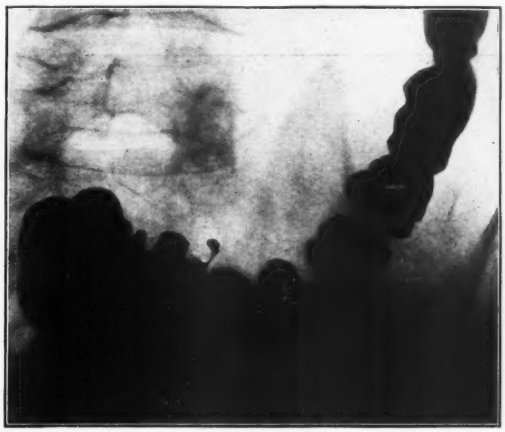

FIGURE IX.
Solitary Diverticulum of Sigmoid.

FIGURE X.
A More Generalized Diverticulosis.

FIGURE XI.
Cystogram—appears normal.

## ILLUSTRATIONS TO THE ARTICLE BY MR. H. B. DEVINE.

FIGURE XII.
Cystogram of same patient as in Figure XI after micturition, showing growth in bladder.

FIGURE XIII.
The pyelogram of very dilated pelvis possibly caused by a neuro-muscular disorder.

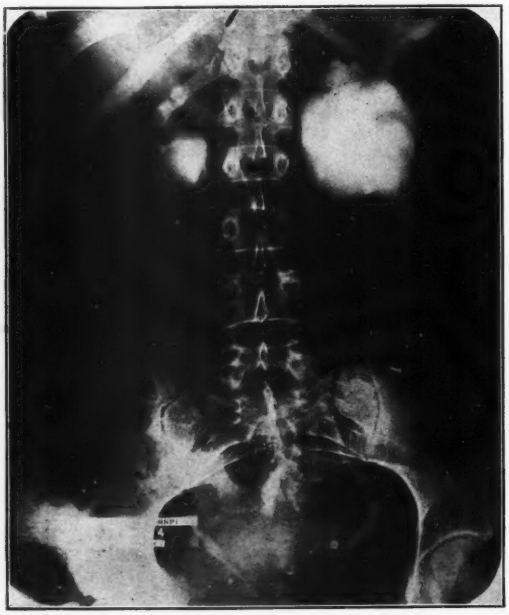

FIGURE XIV.
Bilateral hydronephrosis due to bilateral aberrant artery. There was no clinical indication of the condition on the right side.

## ILLUSTRATIONS TO THE ARTICLE BY DR. JOHN FIDDES AND DR. JOHN A. McLEAN.

FIGURE I.

FIGURE II.

FIGURE III.

FIGURE IV.

## ILLUSTRATIONS TO THE ARTICLE BY DR. JOHN FIDDES AND DR. JOHN A. McLEAN.

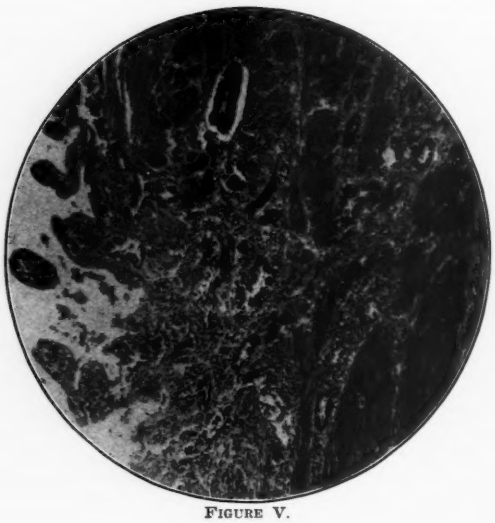

FIGURE V.

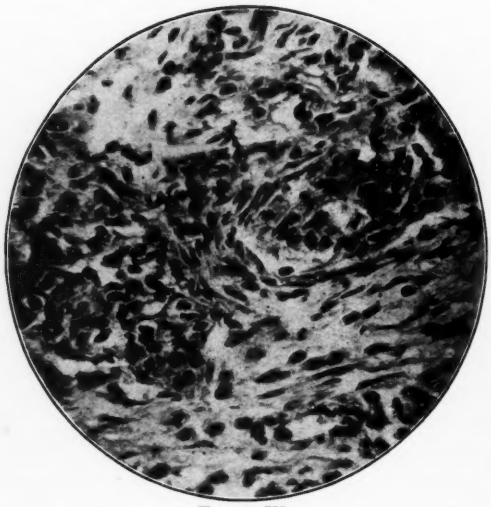

FIGURE VI.

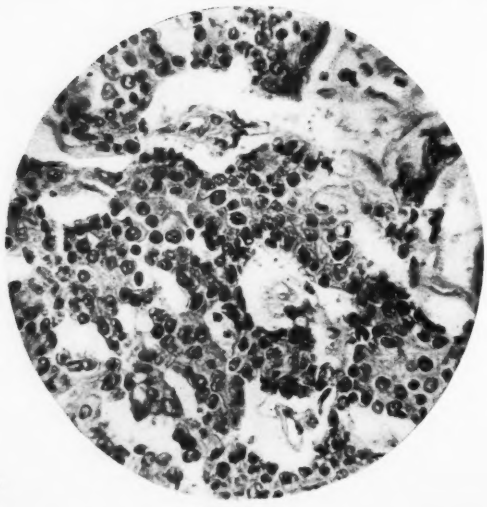

FIGURE VII.

FIGURE VIII.

FIGURE IX.

## ILLUSTRATIONS TO THE ARTICLE BY DR. COTTER HARVEY.

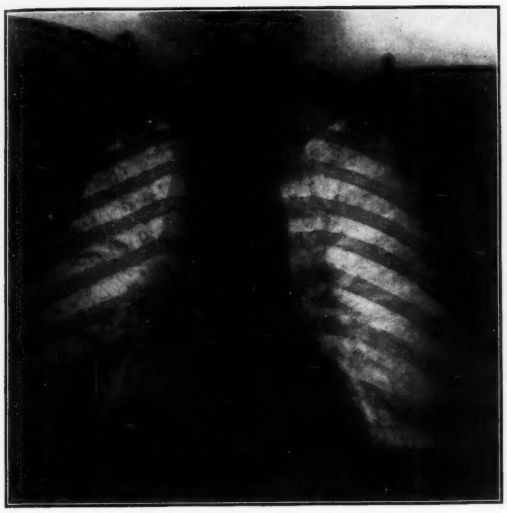

FIGURE I.
Skiagram taken on January 23, showing abscess at base of right lung.

FIGURE II.
Skiagram taken on February 29, showing commencing collapse of right lung.

FIGURE III.
Skiagram taken on March 21, showing complete collapse of right lung.

FIGURE IV.
Skiagram taken on November 21, showing re-expansion of right lung, complete at base, but not yet at apex. Fibrosis at right base and thickened pleura. Compare with Figure I.

## ILLUSTRATIONS TO THE ARTICLE BY DR. R. ANGEL MONEY.

FIGURE I.
Skiagram of injured hand (left) taken eight weeks after injury, showing osteoporosis of all the bones of hand and wrist and an area of chronic osteomyelitis of the stump of the second metacarpal bone.

FIGURE II.
Skiagram of the injured hand taken three weeks after Figure I, showing remarkable increased density in all the bones after massage, active and passive movements, indicating regeneration of bone.

## ILLUSTRATION TO THE ARTICLE BY DR. RICHARD G. S. HARRIS.



FIGURE I.

Many malignant infiltrations of the pylorus are painless. Why is this? The answer is that the carcinoma of the pylorus is generally a slow growing scirrhus and a very slowly applied pressure to gastric tension receptors is not an adequate stimulus to give pain. There may be, however, a crampy pain three or four hours after meals from the hyperperistalsis of the obstructed hypertrophied stomach, but in most cases even this is absent. This particular patient's illness remained undiagnosed because of his youth, till the steady march of his symptoms attracted serious attention and called for an X ray examination. The radiographic diagnosis, perhaps biased by the patient's youth, was a pyloric stenosis, following the scar of an ulcer. The reason given for the diagnosis of scar as against carcinoma was that there was no præpyloric filling defect. Now a scirrhus produces very little filling defect and if the stenosis were due to the scar of an old ulcer, he should have given a clear cut, definite history of a previous ulcer, for pyloric or præpyloric ulcer is always very painful.

As a contrast let me show you a typical history of such an ulcer scar:

K., *ætatis* fifty-eight years, had been ill for seven years. She would be well for three months and ill for about the same period. She complained of a bad burning pain one or two hours after food, particularly after certain foods, such as meat. She vomited and obtained relief. She had not lost weight up to nine months ago. Then her pain ceased, she began to lose her appetite and to get very constipated and now she has lost 13·7 kilograms (two stone) in weight. She has vomited a large quantity every day for the last six months, generally at 9 o'clock. She looks cachetic, ill and malignant. She has no tender spot now, but till nine months ago she had a deep, tender spot in the epigastrium. It should be noticed that her early symptoms were periodic and definitely præpyloric ulcer symptoms.

I would like you also to notice in this history, first the very definite, painful dyspepsia of the præpyloric ulcer, secondly as the ulcer healed and a scar developed two things happened; the pain and tender spot of the ulcer disappeared and the patient manifested almost all the characteristic signs of malignant disease of the stomach, loss of weight, nausea, vomiting, constipation, loss of appetite, all in an old woman of fifty-eight. Here the radiographic diagnosis, perhaps coloured by the patient's age, was malignant stricture. I may mention that both these cases are good examples of what I spoke of in the early part of my lectures, that is, how important it is to make sure in a meticulous way that the clinical symptoms mesh as it were, with the results of the X ray or other special examinations.

### The Syndrome "Inoperable Abdominal Tumour."

What clinically may appear an almost certainly inoperable tumour of the liver or stomach is not always so. For instance, I once saw an old woman, ill for three months, cachectic and wasted, with all the symptoms of malignant disease. She had an irregular cartilaginous hard tumour moving on expiration, which was situated in the left lobe of the liver. There could not be the smallest doubt about the nature of the tumour that it was malignant and that being in the left lobe of the liver it was inoperable. The operation disclosed an irregular, cartilaginous, hard tumour on the left lobe of the liver which, to all present at the operation, appeared to be malignant. Not being quite satisfied with its appearance I inserted a knife and found that it had a hollow centre. Further examination showed that it was the hard cartilaginous puckered wall, twelve millimetres (half an inch) in thickness, of an old degenerated hydatid. A mild suppuration had caused the constitutional signs of malignant disease.

Again, an old woman was sent to me with a somewhat similar history. She had an enormously enlarged liver. The diagnosis was carcinoma of the stomach, with secondary growths in the liver. She was so ill that she could hardly walk and it seemed to be a shame even to think of operating on her. She had no fever and the liver felt as if it contained a malignant growth, although it was not nodular. The skiagram of the stomach revealed no abnormality and, as there was therefore no explanation of the diagnosis of secondary growths in the liver and the condition was somewhat anomalous, I decided on exploration. The operation disclosed a very large, central, mildly suppurating hydatid of the liver.

Quite recently two patients were referred to me with the diagnosis of inoperable malignant disease. Because there was an enormous fixed tumour in the epigastrium, it was thought that operation was useless. Both these patients had what I have frequently seen, an ulcer tumour, that is an enormous inflammatory tumour associated with a penetrating ulcer. At operation you can distinguish these tumours from malignant growths (which, by the way, is very difficult) by the fact that they are so widely and firmly adherent with destruction of planes and for their size are very localized. Notwithstanding all this, there are no outlying indications of malignant disease, such as enlarged glands or small nodules. They have also a different consistence to carcinoma. I have seen conditions like this many times diagnosed and treated at operation by competent surgeons as inoperable malignant disease. Three mistakes were made in connexion with this ulcer tumour I show you the specimen. First, this was considered as an inoperable carcinoma. At a second operation it was thought to be an innocent ulcer tumour and was removed. Microscopic examination showed that actually it was an innocent ulcer which had become malignant.

### Equivocal Gastro-duodenal Ulcer Syndrome.

The equivocal gastro-duodenal ulcer syndrome is an interesting and difficult diagnostic problem. There is a clinical history of ulcer, but no ulcer can be found at operation. The symptoms are often due to a very small ulcer that has been missed at the exploration. I could show you many histories to illustrate this point. These small ulcers are often unrecognizable in the X ray picture. When there is no ulcer, the symptoms are due to the acid

gastritis of Aschoff or to a duodenitis which has the same ætiology. There may be tender points and all the symptoms of a gastric or duodenal ulcer in these latter patients.

As an aid in recognizing these small ulcers by X ray examination, I would like to show you a method of whitewashing the rugæ of the stomach and duodenum which was brought under my notice by Dr. J. O'Sullivan, the Radiologist of St. Vincent's Hospital, Melbourne. Small ulcers are distinguished by the way in which they distort the lines of the rugæ. Figure V illustrates this method. Figure VI demonstrates a very small ulcer distorting the rugæ.

I have here a specimen from a man who was twice explored and his duodenal ulcer was not found because in the first place a history not classical like the usual duodenal ulcer syndrome did not focus attention on duodenal ulcer, and in the second because no duodenal ulcer was obvious from an external examination of the duodenum. The anomalous history and the operative failure to find the ulcer were due to the fact that it was situated on the posterior wall of the duodenum. From the specimen it can be seen that it is a large flat ulcer showing very little tendency to repair. This indolence and its remoteness from the peritoneum are responsible for the difficulty in diagnosis as well as of treatment.

*Clinical Symptoms and No X Ray Signs.*

Although in the skiagram the duodenal cap may appear perfectly normal, yet it may hide a duodenal ulcer. Here is a history.

Mr. J., *ætatis* thirty-four years, had epigastric pain from two to three hours after meals. It was relieved by food or lying down; not relieved by soda or magnesia. He had lost twenty-two kilograms (three and a half stone) in weight, but he had been on a strict diet. He had a deep tender spot over his duodenum. In his skiagram, Figure VI, you will notice what a beautiful normal picture it is, except for one almost unnoticeable spot. You will agree with me that the clinical diagnosis should be duodenal ulcer. Now I know that there is an ulcer in the anterior duodenal wall because I operated on the patient. Which was the more important, the apparently normal cap or the clinical history? We really operated on the clinical history.

I would like now to come to that group of symptoms which suggest duodenal ulcer, to that clinical picture which naturally focuses the observer's mind on the question whether or not duodenal ulcer is present. It is curious that some duodenal ulcers may give scarcely any symptoms at all; perhaps there may be a bad acidity; even the usual deep tender point may not be very obvious. It is not infrequent for these ulcers to be associated with attacks of profound melæna. In the history of one patient it appears that the man never suffered from pain, but had bouts of intense acidity and bad attacks of melæna. He had a very large ulcer of the posterior wall of the duodenum. These ulcers of the posterior wall with rather a latent subjective and objective picture should not be confused with acute duodenal ulcer, which gives neither symptoms nor a deep tender point, but is associated with profound melæna. This condition is akin to acute gastric ulcer and does not require operation; in fact operation is often disastrous. So a keen diagnosis is important.

Many other conditions give duodenal symptoms. I have already said that duodenal ulcer symptoms can be caused by a duodenitis which is generally associated with a very high acid, or they may arise from a painful neuro-muscular incoordination associated with a hypertonic stomach, but the duodenal complex is also a manifestation of duodenal ileus, even to the pain three hours after food, to the deep tender duodenal spot and occasionally to melæna. The dilated duodenum seen in the skiagram will establish the diagnosis. It is important to eliminate duodenal ileus because I am now convinced that it is nearly always a non-surgical neuro-muscular disorder, a functional condition like cardiospasm or similar conditions in the rest of the alimentary canal.

## LECTURE III.

### Abdominal Diagnosis from a Surgical Point of View.

#### Gall Stone Syndrome.

It is true that no difficulty besets a diagnosis in the ordinary case of gall stones, but I have found the greatest difficulty in distinguishing certain phases of gall bladder conditions from duodenal ulcer or even gastric ulcer. I remember one patient who had pain very consistently three hours after meals and had a tender spot over his duodenum. In his history a periodicity like that of a duodenal ulcer was noted and he had been for twelve months under treatment for this disease. He had what appeared to be a deep tender spot over his duodenum. One day he got an attack of severe pain which doubled him up and which created a suspicion that he had gall stones. At operation a gall bladder was found so tightly packed with stones that it could not hold another one. Since that I have seen several patients in whom a tightly packed gall bladder has caused the symptoms of gastric or duodenal ulcer; but every tightly packed gall bladder does not produce these symptoms. In one instance a patient consistently had a violent pain two hours after meals and an attack of vomiting. This man's gastric ulcer-like symptoms were caused by an irregular stone in his common duct which allowed the bile to pass over it and did not produce jaundice. You will find in these cases that very often the radiographic diagnosis of the duodenal cap is equivocal and does not help you, but what will help you is a very accurate and careful investigation of the exact "pressure-axis" of the deep tender spot. Place the finger on the deep tender spot, press against the spine; if it is duodenal ulcer, there should be tenderness. Without displacing the finger turn the pulp up towards the liver; tenderness as the liver comes down with

respiration suggests an inflamed gall bladder which, in these circumstances, is generally nearer the mid-line.

In my experience difficulties in this right hypochondriac region are, first, whether symptoms which are apparently due to gall bladder disease, are not caused by a hepatitis or by chronic "back telling" in a fibrous liver sequent to a failing right heart. These latter, as a rule, give rise to diffuse pain and tenderness. In the second place that it is necessary to find out if the gall bladder is really the cause of a troublesome dyspepsia, which may be painful, but occasionally is only flatulent and painless. In the third place whether there is really a pathological gall bladder basis, a prælecholecystic stage, for an equivocal gall bladder syndrome when gall stones cannot be demonstrated.

In the first I have seen many patients operated on in the belief that they had gall stones. The appearance of the liver in these cases is characteristic; the edge is round and as you would expect, the patient stands the misconceived operation badly.

It is in the second and third condition that I find cholecystography a great diagnostic aid.

*Cholecystography.*

It is difficult to evaluate the diagnostic measure of cholecystography. This has been my experience. Occasionally, where the clinical symptoms were frankly those of gall stones and I used cholecystography as a check and stones did not throw shadows nor did the gall bladder, I operated, nevertheless, on the basis of the clinical observations and always found gall stones. On the other hand, with a most indefinite clinical gall bladder syndrome, cholecystography has been invaluable in incriminating that organ. The reason of this is that it is merely a test of the function of the gall bladder and is specially valuable in those indefinite syndromes where a pathological disturbance of the physiology of the gall bladder is laying the basis for gall stone disease or chronic cholecystitis. Such conditions are the hypertonic and hypotonic gall bladder stasis of Aschoff; the congenitally narrowed or kinked cystic duct. The following interesting experiment quoted by Aschoff illustrates the rationale of cholecystography: The dog has its gall bladder attached to one hepatic duct and it is possible to ligate so that in one case the gall bladder can be left attached to the ducts and the other case the ducts can be ligated so as to exclude the gall bladder. When the gall bladder is left, as it were, in circuit with the ducts, the bile concentrates and becomes thick and viscid because of the concentrating action of the gall bladder. In the other case, when the gall bladder is excluded, the bile becomes more fluid, the pressure rises until secretion stops and the bile becomes quite white. Put shortly, the function of the liver cells and the ducts is to deliver bile secreted from the circulation where it is formed by the reticule endothelial cells. The function of the gall bladder is to regulate the bile pressure and keep it uniform between meals by a process of fairly rapid concentration attained by a resorption by the gall bladder wall of water and some organic salts. To do this the gall bladder wall must be fairly healthy and the bile must be able to enter by the cystic duct. The bile in the gall bladder is six times more concentrated than in the ducts. Another function of the gall bladder is to empty its contents into the duodenum at meal times, especially after a fatty meal. It is possible to estimate these functions by cholecystography.

The oral administration of the dye is very satisfactory and can be carried out without the patient going into hospital. The intravenous gives the best and most certain results but it has, from a practical point of view, obvious disadvantages.

*Interpretation of Cholecystography.*

Cholecystography may yield the following information:

1. If the gall bladder does not fill or is only faintly outlined, (*a*) the capsules have not absorbed, (*b*) there is disease of the gall bladder wall which cannot resorb, (*c*) bile has not entered; the cystic duct may be narrowed, (*d*) bile has not been secreted; there is disease of the liver.

2. There is dilatation or defective emptying, there is hypertonic or hypotonic stasis of Aschoff.

3. There are negative shadows or direct evidence of gall stones.

4. There is deformation of gall bladder; this may be caused by adjacent disease.

5. The situation of gall bladder is abnormal; coordinate this with the clinical symptoms.

6. Note the relation of gall bladder outline to pathological shadows, such as alleged kidney stones.

The following is a history which is a very good example of the peculiar value of cholecystography in prælecholecystitic disease:

Mrs. M. complained of indefinitely localized pain in the right hypochondrium. This pain was associated with nausea and flatulence. She had some diffuse tenderness to the right of the umbilicus. She was operated on in the belief that she had a chronic appendicitis. A long retrocaecal appendix was removed. The surgeon examined the gall bladder and was satisfied that it was normal. All the symptoms returned.

A cholecystograph was then made. Although the gall bladder appeared to be perfectly normal, there was considerable delay in emptying. Such a condition is obviously pathological. A second operation was undertaken because of the results of the cholecystographic examination. Although on external examination the gall bladder appeared normal, it was removed and was subsequently found to be full of small dark pigment stones mixed with semisolid bile. After this operation her anomalous mildly painful dyspepsia cleared up.

*The Surgical Significance of Jaundice.*

As in other regions the crucial point of diagnosis in a patient suffering from jaundice, as in most abdominal diagnoses, is the exclusion or establishment of malignant disease. This is not as easy as it appears at first sight. Incomplete obstruction of the common duct, absence of dilatation of the gall bladder, pain, initial or recurrent, as a rule indicate

gall stones. Progressive history, progressive and green jaundice without remissions or rise of temperature, dilated gall bladder, absence of pain, suggest malignant disease of the pancreas. I should like to stress particularly the importance in diagnosis of the last two points. Cholelithiasis is an innocent disease and can be cured by operation. A phase of it can exactly simulate malignant disease of the pancreas. It is, therefore, a great responsibility to make a diagnosis of this disease in the absence of exploratory operation. The following instructive mistake illustrates very well this point.

The patient, a nurse, aged forty-five years, had been ill for three months with jaundice of the black type, progressive, non-intermittent, with no rise of temperature and no shivers. The symptoms had come on without any pain, so I was told. She had a very enlarged liver. She was being allowed to die without operation because each consultant had made a certain diagnosis of malignant disease of the pancreas and under those circumstances operation was deemed not worth while. In examining her I noticed that although she had a very enlarged liver from a completely obstructed common duct, she had no dilatation of her gall bladder. Now the absence of dilatation of the gall bladder in a carcinoma of the pancreas is very unusual. It is not often that carcinoma of the pancreas occurs with a contracted fibrous gall bladder; I have seen it only in one case. On the other hand the only way that a large stone can get into the common duct is by fibrous contraction and continuous pressure of a diseased gall bladder. The gall bladder with its poor musculature, no matter how hypertrophied, could never push through the cystic duct a stone large enough to block the common duct. Thus it is that a stone in the common duct is always associated with a contracted gall bladder. On carefully reviewing the history of this patient in the light of this fact, I found that there was some slight pain with the onset of her illness.

Operation disclosed a large stone firmly impacted in the ampulla of Vater completely blocking the common duct. The gall bladder was found to be contracted. Notwithstanding the fact that she had what has been called "white bile," regarded as a serious portent, she eventually got well.

Intermittent yellow jaundice, associated with pain, shivers, raised temperature (characteristics of a stone in the common duct) is often caused by a central hydatid of the liver with daughter cysts opening into the bile duct. This condition will give exactly the same symptoms as a stone in the common duct. Here is an instructive mistake in this respect:

For two years a patient had attacks of severe epigastric pain. She had jaundice in one of the attacks. Sometimes she vomited and this eased her pain. Her temperature was 37·8° C. (100° F.) in the attacks between which she was well. In one of the attacks a pleurisy in front of the liver was diagnosed. Two years ago cholecystostomy was performed, but the attacks continued after this. A month ago cholecystectomy was carried out and her appendix was removed. When I saw her she was very ill and jaundiced. Her temperature was 39·5° C. (103° F.). There was tenderness over the scar. Figure VII is the skiagram of her liver. You will notice that the dome of the liver is higher on the right side than it should be. This patient had a central hydatid of the liver.

*The Right Iliac Fossa.*

As you know, the gall bladder can be situated very low in the abdomen and it may give rise to the symptoms of a diseased appendix, but with the aid of cholecystography we are nearly always able to localize the gall bladder and avoid this confusion. The right lower quadrant of the abdomen is a region where many mistakes are made. The majority of appendiceal lesions are straight forward and easily diagnosed, but when diagnoses are made of chronic appendicitis on a tender spot alone or on dyspepsia and a tender spot, it is my experience that many mistakes occur. In addition to the dyspepsia and tender point there should be symptoms corresponding, a history of attacks of what may be regarded as chronic appendicitis. Perhaps because of a generalization that dyspepsia is frequently caused by chronic appendicitis, it has become the custom to remove appendices freely; I think much more often than is necessary. A tender point can often be elicited over a normal caecum. Every patient with duodenal ulcer or gastric ulcer on whom I have operated, has had the appendix removed in the belief that the dyspepsia was caused by the appendix. I find if an appendix is the cause of a dyspepsia, it gives recognizable symptoms and I am sure it is not nearly the factor in the ætiology of dyspepsia that it is supposed to be.

We have all seen a diagnosis of chronic appendicitis followed by appendicectomy when the disease was one of the following: stone in the ureter or kidney, hydronephrosis from an aberrant artery or other causes, tuberculous glands in the mesentery of the appendix, tuberculous peritonitis, mobile ascending colon, prolapsed caecum, abdominal lymphadenitis and so forth. When one sees the enormous number of operations performed, especially in the suburbs of large cities, of appendicectomy and right oophorectomy, one knows at once that either one or other diagnosis is wrong and probably both. Far too much capital, I mean this in every sense of the word, has been made out of appendiceal dyspepsia.

I have just said that the recognition of a diseased appendix is as a rule extremely easy, but I would like to qualify this statement to this extent, that in perhaps 20% the recognition of the diseased appendix, especially if abnormally placed, when it mimics nearly every other disease in the abdominal cavity, is the most difficult and responsible diagnosis that we may have to face.

In my routine diagnostic work, where a patient presents obscure abdominal symptoms, my first object is to localize the appendix or, failing this, the ileo-caecal junction which for all practical purposes bears a sufficiently definite enough relation to the base of the appendix. This can be done by X ray examination. By careful preparation, repeated barium-butter-milk meals, the appendix can be visualized in from 70% to 80% of patients.

The usual appendiceal syndrome is epigastric or umbilical pain, followed by pain over McBurney's point, then sickness or vomiting, then constipation, associated with the rigidity and tender point, all in this order. If sickness or vomiting precedes the

pain, be on your guard. The illness is generally not appendicitis. Remember that in a fat patient the tenderness is less than it should be. If the onset of the pain is intense, you may be sure the patient is in for a bad attack of appendicitis and if it clears up suddenly in twelve to twenty-four hours, that does not show that it has got better, but that the patient is in the "dangerous relief interval" of a fulminating appendicitis. Gangrene or perforation has given the sudden relief from pain and it is at this stage that there is no fever and very often no increase in the pulse rate. To the experienced eye the appearance of the patient at once indicates what has happened. Many appendiceal catastrophes have occurred because this fact has not been recognized.

As I am giving, under the ægis of the College of Surgeons, a lecture entitled "Difficult Appendicectomy," I understand to the same audience, I shall postpone what I have to say on atypical appendiceal syndromes till then.

#### Symptoms and Signs in the Umbilical Region.

A malignant tumour of the pancreas is not always associated with jaundice. Carcinoma of the body or tail of the pancreas begets mild dyspeptic symptoms, anæmia, loss of weight, a picture which is indistinguishable from cancer of the body of the stomach. In fact it is nearly always confused with this. You find the patient has a tumour in the umbilical region and these symptoms and you send him for X ray examination. You wonder why the radiographer says there is no filling defect in the stomach to account for the symptoms. You infer the diagnosis of gastric carcinoma and find pancreatic carcinoma.

A man had such a symptom complex. The X ray picture was negative. There was a tumour in the umbilical region. He had no pain. The condition was diagnosed as carcinoma of the stomach. He had a carcinoma of the body of the pancreas.

Now here is the history of another patient and I would like you to contrast this history with the previous one.

R., aged sixty-two years, had had dyspepsia for eleven years. Four months age he began to get pain in the upper part of the abdomen and in the right hypochondrium. Meals made no difference to the pain. One month ago he developed excruciating pains in the epigastrium which caused him to double up and to roll about on the floor. Large doses of morphine did not relieve the pain. The attacks lasted from ten minutes to hours and he got an attack every day. He had no tumour and an indefinite tender spot over his pancreas. It was impossible to make a diagnosis. Operation disclosed scirrhous carcinoma of the middle of the pancreas.

Here was a carcinoma in one patient causing excruciating pain in the region of the pancreas with no palpable tumour and in another patient a tumour in the very same situation with no pain.

Apart from the acute and mildly acute types of pancreatitis there are subacute attacks which give rise to a syndrome of central abdominal symptoms, rather difficult to diagnose. Here is such a history which illustrates what I mean. A woman, aged thirty-three years, had suffered from previous attacks of epigastric pain. She was seized on Monday with moderately severe pain just above the umbilicus, associated with continuous vomiting. On Wednesday the pain and vomiting were still present and her temperature was 38·9° C. (102° F.) and her pulse rate was eighty. She had moderate rigidity of the upper part of the abdominal wall, diffused central abdominal tenderness and also appendiceal tenderness. At operation pancreatitis with peritoneal fat necrosis was found, a large plaque under McBurney's point accounting for the appendiceal tenderness.

#### Diagnostic Problems of the Left Iliac Fossa.

Personally I find the left iliac fossa the most difficult region of the abdomen. Affections of this region are associated with some obscure syndromes such as ill health with mild abdominal distention, ill defined pains, with constipation and passage of mucus. Is this a beginning sigmoid carcinoma or is it just a sigmoiditis, perisigmoiditis or diverticulitis, the evidence of which we often see in routine exploration of the stomach?

A scirrhous carcinoma produces obstruction and no palpable tumour; a fungating carcinoma produces a palpable tumour and no obstruction; microscopic examination of the fæces in the latter always gives some evidence, such as pus or blood, because these fungating carcinomata always ulcerate early. This evidence, combined with a filling defect, usually easily seen with the clysma and the fact that because they have no obstruction the patients come late and have a palpable tumour, means that as a rule such a condition does not give much trouble in diagnosis. On the other hand the scirrhous type of carcinoma of the sigmoid, although it produces symptoms of obstruction early, with distention and colicky pains, is difficult to diagnose early because it gives an indefinite filling defect. It is most important in this latter type to make an early diagnosis, because operation is particularly beneficial for a malignant colon growth. The following is a characteristic history of an early scirrhus:

A woman, aged sixty-seven years, complained of pain, wave-like and transient, very severe when she had a motion, worse half an hour after meals, situated in the left iliac fossa, rolling down to the suprapubic region. Constipation had been much worse during the last six months. She had lost a little weight and looked fairly well. She had no palpable tumour. The clysma was partly held up at a point in the pelvic colon at which a small filling defect could be seen (see Figure VIII). The diffuse edges of the defect can be seen.

When the tumour is situated too high for detection with the sigmoidoscope, great help is obtained by watching the clysma go through the affected part, that is, having focused attention on a suspicious part, the examination should be repeated and the part carefully studied with the aid of the radiographic screen.

An innocent sigmoid condition is often mistaken and operated on for carcinoma.

A man complained of constant dull pain in the left iliac fossa which he eased by taking opening medicine. Six weeks ago he had an attack of very severe pain in this region and vomited large quantities of fluid. His abdomen

swelled up and the doctor had great difficulty in getting his bowels open with enemata. There was tenderness in the left iliac fossa and a definite resistance, an apparent tumour. There was some difficulty in the passage of the clysma through this point and he had occult blood in his fæces. In Figure IX the little diverticulum can be seen. This patient's condition was diagnosed as carcinoma of the sigmoid, because he had an attack of bowel obstruction requiring repeated enemata for its relief, a tender point, slight reflex rigidity and a palpable tumour in the left iliac fossa. The diverticulum had become inflamed and brought about this syndrome.

This condition is a much more frequent cause of left iliac fossa symptoms than is generally supposed. Figure X is the reproduction of a skiagram in which the general distribution of this diverticulosis can be seen. There is a more acute phase of this disease which is illustrated by the following history: A patient presented himself because he was suffering from pain and tenderness and tumour of the left iliac fossa. The tumour was obviously an abscess and was opened. A sinus formed and this skiagram reveals the injected sinus passing through a diverticulum into the sigmoid. The abscess was due to a rupture of one of these diverticula. In relation to this diverticulosis it is important to remember that in exploring the abdomen you have to be very careful lest you knock off any of these little diverticula. If you do, you will find a quietly developing peritonitis the cause of which you will never suspect.

The following is a very interesting and instructive history in relation to everyday diagnosis in this region.

The patient, aged fifty years, complained that she had been constipated during the last six months; latterly it had become so severe that it was with the greatest difficulty that she could get her bowels open with an enema. She complained of colicky pains especially over the left lower part of the abdomen. These were associated with distention. She got relief when her bowels opened. There was tenderness over the left iliac fossa. The skiagram revealed obstruction of the sigmoid. There was no pus or blood in her stool and nothing abnormal could be seen with the sigmoidoscope in the lumen of the bowel. At operation a large absolutely inoperable growth was found which was thought to be carcinoma. She was given deep radiotherapy and at a second operation removal was attempted. With the greatest difficulty, the ureters having to be dissected out of the mass, the growth was removed. I was surprised to find that it was a diverticulitis.

This is a more advanced case than the one I have just mentioned. Here many diverticula had become inflamed and joined into a solid inflammatory mass. It is said that diverticulitis does not cause complete obstruction and never hæmorrhages into the lumen, that it is more tender than a carcinoma, but there is no doubt that they are extremely difficult to distinguish from a carcinoma, even at the operation.

Not long ago I operated on a patient who had been previously subjected to an operation for a carcinoma and complete obstruction. A colostomy had been done, the surgeon declared that the man had inoperable carcinoma. Something made me suspicious and I decided to explore his abdomen. The mass when removed proved to be a diverticulitis.

### Genito-urinary Disease.

My remarks on abdominal diagnosis would be incomplete without some reference to genito-urinary diagnosis. I can make only a few generalizations. One is that in this region, more than anywhere else, a routine examination is necessary. One must not jump to conclusions. A patient had a painless hæmaturia, cystoscopic examination revealed a sessile growth on the bladder; this was removed. The hæmaturia still continued. Ureteral catheterization proved that the bleeding was coming from the left kidney. Operation disclosed a hypernephroma. That was ten years ago and the patient is still alive.

In another case of hæmaturia the prostate was found to be enlarged. Apparently the bleeding came from this organ. (Probably this was due to trauma of the cystoscope.) The prostate was removed. The hæmaturia still continued, but subsequent examination showed that the blood was issuing from the right ureter. The patient had a growth on the right kidney. A patient for years complained of pain in her right kidney. A stone in the right kidney was discovered by X ray examination. She refused surgical treatment. Four months ago she began to get pain and frequency of micturition. This increased, hæmaturia gradually developed with great pain at the end of micturition and the pain in the bladder became continuous. She had no pyuria. Clinically this was a picture of malignant disease of the bladder, yet she was watched for four months in the belief that she was passing the stone through the ureter or that it was in her bladder. The carcinoma was removed from her bladder by an extensive partial cystectomy.

These patients are difficult to examine with a cystoscope because their bladders are so small and it is difficult to get a view of these big malignant growths in order to see whether you can operate on them or not.

Here is a method suggested to me by Dr. O'Sullivan, Radiologist at St. Vincent's Hospital, Melbourne, which I find very helpful. The bladder is filled with 30% sodium iodide. Figure XI represents the usual cystogram. When the patient empties his bladder, the sodium iodide adheres to the irregular surface of the growth. The actual shape and size and situation of the growth can be seen in Figure XII. A careful diagnosis of these conditions is operatively useful and often curative.

The second point in genito-urinary diagnosis which I wish to make, is that you must always be certain as to whether the disease that you observe, is primary or secondary. If it is secondary, you must always be careful to find the primary condition. This, as you know, obtains in connexion with tuberculosis of the bladder for the primary focus is generally in one or other kidney.

In the case I mentioned previously the sessile growth on the bladder was secondary; the primary was the hypernephroma in the kidney which I failed to find at the first examination and produced the secondary bladder tumour by the implantation of

hypernephroma cells in the bladder wall. Again, to illustrate this principle, I have a skiagram here which shows many small stones in the kidney. On the affected side there is obviously a dilated pelvis and the multiple stones are much lower in relation to the lower poles of the kidney than they should be, indicating that they are lying in probably a dilated pelvis. Here a pyelogram is indicated and will show that the pelvis is dilated. The stones in this case are secondary. A very careful examination of the pelvo-ureteral junction revealed a fibrous stricture. This was dilated; unless the condition had been recognized from the pyelogram, it might have been assumed one of the small stones had temporarily blocked the ureter and had dropped back.

Again, a large diverticulum with small opening is discovered with the cystoscope. It is hailed with delight as the cause of the patient's obscure symptoms, symptoms which have baffled many examiners. The diverticulum is removed, but, alas, it was only secondary. Many of the patient's symptoms still persist, the suprapubic wound closed unkindly because the diverticulum is really caused by a neurological defect in the emptying of the bladder.

Always be sure that the other kidney is sound.

Here is the history of T. who complained of pain in the right kidney region, and on examination a tumour was discovered. She had pus in urine. Figure XIII is a reproduction of her pyelogram.

It was found at the operation that she had a greatly dilated pelvis although there was no obstruction at the outlet. The obstruction was apparently due to a neurological defect (? bilateral) in emptying caused by an incoordination of the muscle, for strong peristaltic waves could be seen running to a point near the pelvo-ureteral junction. Urine flowed freely if a cannula was inserted and from the pelvo-ureteral junction when the kidney was removed from the body and held up; but not while the kidney was connected to the body and the muscle was alive.

Mrs. C., *ætatis* thirty-four years, had a ten years' history of kidney trouble. She had a gynæcological operation for her pain, with no effect. She complains of a dull ache behind the left kidney, which sometimes becomes a very terrible pain and then subsides into a dull ache till she gets another severe pain. She is getting worse lately and vomits when the pain is severe. There is no pain on the right side and there was no suspicion of disease on the right side. A double pyelogram (see Figure XIV) reveals a dilated pelvis on both sides. There is an aberrant artery on both sides. Without this knowledge, I would have removed the kidney on the diseased side.

In conclusion I must ask you to bear in mind that these lectures are the angle of view, the experiences of one surgeon—myself—and as such must be assessed and assimilated.

## CARCINOMATOSIS OF THE PERITONEUM.

By John Fiddes, M.D.,
and
John A. McLean, M.B., B.S.
(From the Pathological Laboratory, Baker Medical Research Institute, Alfred Hospital, Prahran.)

Widespread tumour growth, confined to the lining visceral and parietal layers of the great serous cavities without obvious primary growth elsewhere, has been described by various authorities as being endothelioma or mesothelioma.

It is well known that malignant tumours may by metastases give rise to diffuse subserous growths greatly resembling those described as primary in the serous membranes. For example, cancer of the breast may secondarily invade the pleura and by lymphatics of the falciform ligament spread to the peritoneum. Carcinoma of the alimentary tract, especially so-called mucoid (or colloid) cancer, can cause a diffuse tumour growth of the peritoneum, but probably the most common origin of peritoneal invasion is ovarian cystadeno-carcinoma.

We are describing four cases of malignant invasion of the peritoneum which we consider to be of interest clinically on account of the difficulty of accurate diagnosis and pathologically on account of the resemblance to endothelioma of the peritoneum as described by various observers.

### Case I.

*Clinical Notes.*

A female patient, aged eighty-one, complained of dyspnœa and loss of weight for six months and the abdomen was noticed to be swollen for some time prior to admission. On examination definite enlargement of the abdomen was found with lateral dulness and fluid thrill. A mass was palpable in the upper part of the abdomen. Paracentesis was performed and 6·2 litres (eleven pints) of clear fluid removed.

*Autopsy.*

The abdomen contained a great quantity of clear yellow fluid. The intestines and mesentery were covered with innumerable small, translucent, waxy-looking nodules (Figure I). The great omentum appeared as an elongated cylindrical tumour mass extending across the abdomen just below the transverse colon. The parietal peritoneum in the pelvis showed a diffuse white thickening of firm consistency. Extending up the abdominal wall this thick white layer became thinned out into many interlacing strands between which the peritoneum appeared normal (Figure II).

The Fallopian tubes and ovaries could not be readily identified owing to the tumour-like thickening of the peritoneum. On the left side there was a single ovarian cyst about 3·75 centimetres (one and a half inches) in diameter and containing thick brown fluid. An incision through the mass on the right side revealed a small calcified ball enclosed by fibrous tissue. A careful examination of the alimentary tract and other organs revealed no abnormality that could be related to the tumour condition.

*Microscopical Appearances.*

The tumour cells contained a relatively large oval or rounded vesicular nucleus rich in chromatin. The cytoplasm, faintly granular, was neutrophile. The cell outline was well marked, giving a mosaic appearance to a group of cells. Mixed with these cells were others with more deeply staining and indefinitely shaped nuclei. Masses of tumour cells were embedded in a fairly cellular fibrous stroma (Figure VI). In some parts the cellular nature of the stroma resembled sarcoma. All subperitoneal organs had this covering of tumour, the relative amount of stroma to tumour varying in different parts. There was some infiltration downwards into the viscera, but no metastases outside the peritoneal cavity were found.

### Case II.

*Clinical Notes.*

A female patient, aged twenty-eight, complained of increasing distension of the abdomen, weakness and breathlessness on exertion following an operation performed two or three months before admission. At operation a large malignant ovarian cyst was removed which burst during

removal. The patient's condition gradually became worse until death occurred about four weeks after admission.

*Autopsy.*

On opening the abdomen the peritoneum presented an opaque grey appearance, due to tumour growth. Here and there, where the growth was more scanty, it took the form of nodules and plaques.

The liver was adherent to the diaphragm which was thickened and infiltrated with tumour growth. On section the liver manifested a well demarcated greyish white border nearly six millimetres (a quarter of an inch) thick, contrasting with the pale greyish pink of the liver substance.

The tumour growth had caused irritation of the diaphragmatic pleura, binding down the lower lobe of the left lung by firm adhesions. The lung itself was not infiltrated by the tumour. The stomach and part of the small intestine were adhering to each other and to the parietal peritoneum; elsewhere the peritoneal surface was free and smooth. There was a greater extent of thickening in the pelvis, especially in the region of the right ovary. One ovary could be distinguished in the tumour mass; the other could not be found. The growth extended peritoneally over the uterus and broad ligament and posterior surface of the bladder. The spleen was moderately enlarged, firm and congested. Its peritoneal investment appeared as an almost white border of tumour growth, four millimetres (one-sixth of an inch) thick, making marked contrast to the deep red colour of the splenic substance underneath. No extension of the tumour outside the peritoneal cavity was found.

*Microscopical Appearances.*

Microscopically the tumour cells invading the peritoneum had a finely granular eosinophilic cytoplasm with large vesicular nuclei and were arranged in masses the outer cell layer of which tended to be arranged in columnar fashion (Figure VII). In addition to surface spread over the liver there was infiltration of tumour cells along the portal canals. These cell masses preserved those same characteristics. All the organs covered by the peritoneum showed some infiltration from the surface growth, particularly in the uterus where the metastases had spread deeply about half way through the muscular coat.

**Case III.**

*Clinical Notes.*

A female patient, aged sixty-five, complained of obstinate constipation and recently of occasional vomiting and generalized abdominal pain. On examination the abdomen was slightly distended, with generalized tenderness.

*Autopsy.*

On opening the abdomen there was a large quantity of clear yellow fluid. The parietal peritoneum was thickened and white and the visceral peritoneum manifested white plaques and small nodules. There was a thickened area on the lesser curvature of the stomach just above the pylorus. On opening the stomach the mucous membrane over this area was apparently normal, except for slight contraction at the centre. There was no enlargement of the lymphatic glands of the lesser curvature or of the hepatic glands. The peritoneum of the greater and lesser omentum and mesentery was thickened and contracted. Section of the duodenum and intestines revealed no abnormality, except the peritoneal changes. The peritoneum over the liver and spleen was greatly thickened with new growth. The bladder, the uterus and adnexa were apparently normal, except for the thickening of the peritoneum. The other organs presented no gross abnormalities and no evidence of metastases or tumour formation was detected elsewhere.

*Microscopical Appearances.*

The tumour was seen taking origin from the glandular epithelium of the pyloric end of the stomach, breaking up the muscular mucosa and invading the submucosa and muscular coats. In the submucosa the tumour cells were seen in large numbers, lying singly in the connective tissue. The tumour cells were scattered and discrete with little tendency to lie in apposition. They contained an irregularly oval, deeply staining nucleus and strongly eosinophile cytoplasm and many of them were vacuolated. In the broken *muscularis mucosæ* many lymphocytes were present and also a few plasma cells. The tumour was continuous with the subserous infiltration elsewhere. The subperitoneal coat of the large and small intestines was infiltrated more thickly (Figure III), the cells tending to group together and connective tissue stroma was more scanty (Figure VIII). The same condition was found on the surface of all subperitoneal viscera (Figure IV), the tumour tending to remain superficial and to infiltrate the underlying organs only to a small extent.

**Case IV.**

*Clinical Notes.*

A female patient, aged sixty-three, complained of weakness and loss of weight. These symptoms had been present for about nine months and during the two months previous to admission her abdomen had become distended. She suffered from flatulence, but no other symptoms of indigestion were noted, except constipation. On examination of the abdomen there was considerable distension with a dull note to percussion anteriorly and a questionable fluid thrill.

*Autopsy.*

The abdomen contained a large quantity of straw-coloured fluid. The parietal peritoneum was greatly thickened, forming a tough white lining to the abdomen. This thickening was most pronounced over the diaphragm and the involvement was least marked in the pelvis. The visceral peritoneum of the small intestine and mesentery showed occasional plaque-like thickenings and opacities. The great omentum was thickened by the tumour formation and adherent to the anterior abdominal wall and gall bladder. The liver (weight 1,868 grammes or 66 ounces) was pale in colour and of soft consistency. Examination of a frozen section stained with Sudan-III revealed pronounced fatty degeneration of the hepatic cells. There were two or three small firm white nodules in the liver substance adjacent to the gall bladder. The gall bladder was embedded in peritoneal adhesions which were greatly thickened by tumour formation and in addition it contained six large facetted gall stones. The mucous membrane presented no macroscopical abnormality. The cystic duct and the bile ducts were apparently normal. The abdominal lymphatic glands were not enlarged and no abnormality was detected in the other abdominal organs, except the uterus which contained several myomata. The tumour condition had spread through the diaphragm to the pleura which showed several white plaques with secondary congestion. No pleural effusion was present.

*Microscopical Appearances.*

The surface mucosa of the gall bladder had been shed while the deeper parts showed atypical proliferation of the glands merging into columnar cell carcinoma which invaded the muscular coat (Figure V) and outer subserous coat, the latter being greatly thickened with fibrous tissue. The liver in the gall bladder region showed extensive fibrosis, leaving islands of liver cells in various degrees of fatty degeneration and atrophy. This fibrotic tissue was extensively invaded by the tumour which had a scirrhous arrangement in the more dense parts, and elsewhere retained a tubular structure. The small intestine, ovaries and abdominal lymphatic glands showed infiltration of peritoneal surface with carcinoma which everywhere showed tubular arrangement (Figure IX).

**Discussion.**

The peculiar naked eye appearance of these tumour conditions of the peritoneum, with their diffuse spread over the subperitoneal viscera and

parietes, inclines the observer to consider the possibility of their origin from the cells of the covering serosa. It must be remembered that the lining cells of the pleura and peritoneum are not truly endothelial. Woollard[1] in a recent Listerian oration published in The Medical Journal of Australia states:

> Though we usually talk about the endothelium of the peritoneum, the pleura and the cerebro-spinal spaces *et cetera*, yet strictly speaking the name mesothelium should be reserved for these, for we know that cells lining these spaces have a different origin and these cells different functions from the endothelial cells of vascular and lymphatic systems.

Ewing,[2] in his latest edition of "Neoplastic Diseases," quotes Rokitansky's description of primary cancers of the peritoneum. These, he states, are composed of multiple translucent nodules lying beneath the serosa and covering many organs. Many observers have described primary endothelioma of the serous membranes. Miller and Wynn[3] describe the origin of a tumour which they call primary endothelioma, from the lining cells of the peritoneum, observing all stages of their proliferation up to large nodules. They state:

> An interesting point about the malignancy of our tumour is that, although it infiltrated tissues and organs, there is no infection of the lymphatic glands and no metastatic growths are seen. There is considerable amount of infiltration of subperitoneal tissues, notably in the diaphragm, and also in the submucous coats of the intestines *et cetera*.

It will be seen from our description that all our four tumours resemble each other closely in naked eye appearance. The diffuse spread of tumour growth along the parietal peritoneum (Figure II), the multiple nodules and plaques on the peritoneal surface of omentum, mesentery and alimentary tract (Figure I) are common to all, though in some the spread is more diffuse than nodular, indicating a later stage. This method of spread has a close resemblance to Rokitansky's description of endothelial carcinoma.

Microscopically, however, there were distinct differences. In Cases II and IV the carcinomatous nature is seen at a glance. In Case II the cells, with large rounded vesicular nuclei and granular protoplasm, grouped themselves in masses, with an apparent intercellular material. The cells bordering the stroma showed a definite tendency to arrange themselves in columnar fashion so that their nuclei were arranged in definite line and not in the irregular, disorderly fashion so often seen in malignant tumours. In Case IV the tumour cells appeared to maintain, even though atypically, the tubular arrangement of the glands of the gall bladder mucous membrane from which we traced the origin of the tumour. It was remarkable that even in the distant ovary invasion of the superficial stroma showed the same definite tubular arrangement. White and Brunton[4] report a primary carcinoma of the peritoneum similar to this, but examination of the gall bladder is not mentioned, although other organs, including the liver, were reported as examined. Our metastases in the liver also show similar structure.

In Cases I and III the microscopical appearance might easily apply to primary endotheliomata as described by various authors. In each case the cells were more scattered and the arrangement less orderly and there was a more intimate relation between them and the surrounding connective tissue. There was a difference, however, in cell structure. In Case I the deep staining oval somewhat vesicular nucleus was surrounded by a cytoplasm staining very faintly and almost devoid of granules. It had a very distinct cell membrane which showed best when two cells lay side by side. The cell membrane gave a mosaic appearance to a group. Case III, with a similar nucleus, had a more granular and eosinophilic cytoplasm and the cell membrane was practically invisible. Both I and III were accompanied by much fibrous tissue, giving them in places a scirrhus-like appearance.

Three of the cases were in patients over sixty years of age and in these there was preponderance of fibrous tissue accompanying the spread of the tumour. This would be expected in the slower growth of the neoplasm in aged persons. One in a young person was predominantly cellular and although of shorter duration had extended deeply into the subjacent organs. By the law of homologous growth it is easy to understand why tumours of the ovary should choose the peritoneal route for their spread; but it is less obvious why such tumours as arise from the gall bladder and pyloric region of the stomach should act similarly. In most cases the latter tumours tend rather to spread by lymphatics to the liver and other organs, only locally, if at all, involving the peritoneum.

The adenomatous cystic tumours of the ovary are generally considered to have origin from the germinal layer of the ovary or from Pflueger's tubes which Walthard has demonstrated as persisting as downward growths in the adult. These, like the peritoneal cells, are of mesodermic origin.

In Case II there is little doubt that the spread of the tumour had its origin in implantation from the ruptured cystadeno-carcinoma. An interesting point about these tumours is their method of spread and their distinct tendency to early and extensive peritoneal involvement, only later involving lymphatic glands and organs external to the peritoneum.

This method of growth resembles closely that described for primary endothelial carcinomata. Two of our tumours could easily have been mistaken for primary growth of the peritoneum had not a careful search been made for a source of origin. In Case I the actual origin could not be verified; but the density of the tumour mass in the pelvis and the impossibility of dissecting out and identifying the right ovary appeared to us a likely evidence of its origin there.

We consider that no tumour of the serous cavities should be called primary endothelioma, mesothelioma or carcinoma until very careful scrutiny is made of all possible sources of origin. It is extremely likely that less and less will be heard of

the terms endothelioma and mesothelioma of the peritoneum in the future, as better methods of investigation are brought into being.

### Summary.

1. Four cases of peritoneal carcinomatosis are described.

2. In one case the source of the growth was a ruptured ovarian cyst. In the other three cases the origin was not obvious until confirmed by microscopical examination.

3. The resemblance of these tumours to so-called primary endothelioma of the peritoneum is discussed.

4. We agree with many authorities that primary carcinoma of the peritoneum is a very rare condition and that the great majority of cases reported as such have a primary focus elsewhere which, being insignificant, has been overlooked.

### References.

(1) H. H. Woollard: "Listerian Oration," The Medical Journal of Australia, August 4, 1928, page 134.

(2) Ewing: "Neoplastic Diseases," Third Edition, 1928, page 349.

(3) James Miller and William H. Wynn: *Journal of Pathology and Bacteriology*, Volume XII, 1908, page 267.

(4) C. Powell White and C. E. Brunton: "Some Uncommon Tumours," *Journal of Pathology and Bacteriology*, Volume XXX, 1927, page 313.

## Reports of Cases.

### SUPPURATING HYDATID DISEASE OF THE LUNG CURED BY ARTIFICIAL PNEUMOTHORAX.

By Cotter Harvey, M.B., Ch.M. (Sydney), T.D.D. (Wales),
*Honorary Assistant Physician, Royal Prince Alfred Hospital; Honorary Physician-in-Charge, Clinic for Pulmonary Diseases, Royal North Shore Hospital, Sydney.*

The following case has some features of interest. Mrs. A.W., aged thirty-two years, was admitted to the Royal North Shore Hospital on January 21, 1928, complaining of cough and much offensive sputum, with frequent night sweats, for six weeks. She had spent practically all her life in the country and had had no previous illnesses of any sort. On admission she had a temperature of 38·1° C. (100·5° F.) and pulse rate of 94 per minute. The report on a skiagram of her chest taken on January 23 (Figure I) was: "Right basal abscess or extensive bronchiectasis suggested." For the next four weeks she coughed up daily 180 to 240 cubic centimetres (six to eight ounces) of purulent but not offensive sputum and her temperature rose nightly to round about 37·8° C. (100° F.). Her general condition remained fairly good. She was seen by the writer in consultation on February 12, at which time there were signs strongly suggesting a cavity at the base of the right lung. A diagnosis of pulmonary abscess was made and a report asked for on the accessory nasal sinuses and, in view of her country life, tests for hydatid disease were also carried out. All sinuses were reported free from disease. The blood count revealed 22,000 leucocytes per cubic millimetre, with 80% polymorphonuclear cells and 2% eosinophile cells. The Casoni dermal reaction was present and a positive response to the complement fixation test for hydatid disease was obtained with the patient's serum. This was confirmed by the pathologist at the Board of Health. No evidence of hydatid disease of the liver was found clinically or by X rays. A skiagram of the patient's chest, taken on February 20, revealed no change. Frequent examination of sputa failed to reveal any tubercle bacilli or any hydatid elements.

At this juncture the surgeon expressed his desire to perform a thoracotomy, but he agreed to await an attempt to cause collapse of the lung by artificial pneumothorax.

Artificial pneumothorax was commenced on February 24. A satisfactory reading was obtained and 750 cubic centimetres of air were introduced into the right pleural cavity. The sputum fell in amount to 90 cubic centimetres (three ounces) daily and became gradually less and also less purulent. Refills were continued and Figure II shows the lung collapsing after the second refill. After the fourth refill the collapse was complete, no adhesions being present and the mediastinum was not displaced. This is well shown in the third picture, taken on March 15. The patient's condition had been undisturbed by the treatment, her temperature was now remaining at normal and the sputum had been reduced to less than thirty cubic centimetres (one ounce) daily, frothy and mucoid in consistence and no longer purulent.

She was discharged in excellent condition on March 29, 1928, and continued to attend regularly for refills as an out-patient. As she remained in perfect health, the last fill was given in September and the lung has now (December) fully expanded, with no return of symptoms. The fourth picture, taken on November 21, 1928, when the lung had almost completely expanded, shows an entire clearing of the mottling and the rounded shadow at the right base. There is evidence of some fibrosis in the lung and some pleural thickening, but healing of the process appears to be complete.

I am indebted to Dr. R. S. E. Todd, under whose care this patient originally came, for permission to publish these notes.

### Comment.

Though no hydatid elements were found in the sputum, there seems little doubt that a suppurative process had become engrafted on an old hydatid cyst in the right lung. This may have been secondary to a liver cyst; evidence of this was lacking.

The main object, however, in publishing this case is to draw attention to artificial pneumothorax as a conservative method of treating chronic pulmonary suppuration. Had this patient been subjected to a pneumotomy, in addition to the risks attendant on a major surgical operation on the thorax, there would have resulted a sinus, persisting for several months, with its debilitating effect on the patient's health. Here no shock whatever was felt by the patient. Improvement commenced from the first injection of air. No special nursing care or frequent changing of dirty dressings was required; from the hospital point of view these advantages do not require stressing.

The patient was able to leave hospital cured in one month and to resume her domestic duties forthwith.

The sequel to surgical interference would have been a greatly damaged lung; perhaps a permanent fistula or a cirrhosed lung, with mediastinal displacement and its embarrassing consequences.

It may be objected that this is an exceptional case and that usually pleural adhesions will prevent the induction of artificial pneumothorax. While not entirely admitting the truth of this contention, a reply can be given. First, let the physician attempt to introduce air into the pleural cavity. If this fails, the surgeon can carry out his pneumotomy in one stage with the assurance of an adherent pleura. Except in those comparatively rare cases in which there is no evidence of bronchial drainage, it would appear to be a barbarous and irrational procedure for the surgeon to plug gauze into the pleural cavity with the object of creating adhesions before opening into the lung, thereby destroying any hope of conservative treatment.

To the objection that collapse therapy may prevent instead of facilitate drainage, it is surely arguable that a

bronchus is almost as rigid as a rubber drain and mere air pressure could not occlude it. In six cases of chronic pulmonary suppuration, a small number admittedly, treated thus, no blocking of bronchus has resulted nor any septic "pocketing" symptoms.

It may be noted that Harold Dew in his recent book on hydatid disease mentions the possibility of cure of suppurating hydatid disease of the lung by artificial pneumothorax. To the writer's knowledge, no case has yet been reported of the adoption of this form of treatment in pulmonary hydatid disease.

### Summary.

1. A case of pulmonary suppuration of hydatid origin is shown to have been cured by the induction of artificial pneumothorax.

2. The advantage is stressed of this method of treatment over surgical procedures in chronic lung-sepsis.

3. It is urged that in any case of chronic pulmonary suppuration, careful consideration be given to the possibility of a conservative cure by artificial pneumothorax before resorting to more drastic surgical measures.

## OSTEOPOROSIS OF THE HAND FOLLOWING INJURY.[1]

By R. ANGEL MONEY, M.C., M.B., Ch.M.,
*Honorary Assistant Surgeon, Royal Prince Alfred Hospital, Camperdown, New South Wales.*

THE patient, a male, *ætatis* fifty-three years, injured his left hand early in March, 1928, with an axe. The wound opened his second metacarpal-phalangeal joint and became septic, causing arthritis of that joint and osteo-myelitis of the second metacarpal bone. This necessitated amputation of about half of that bone together with the index finger. The stump became involved later and the whole hand, wrist and lower portion of the forearm became swollen and subacutely inflamed.

The septic process gradually settled down under appropriate treatment with baths, foments *et cetera*, during which time the hand was kept at rest on a splint. A small area of inflammation remained, however, around the stump of the second metacarpal bone, where two small sequestræ developed.

When first seen on May 21, 1928, he complained of a lot of pain in his hand and inability to move the fingers and the wrist. His hand was still œdematous and the skin had an atrophic appearance. Practically no active movements were possible and creaking adhesions were present in all the joints of the hand and wrist, limiting all passive movements to a pronounced degree. A small area of redness and tenderness was present in the centre of the scar over the stump of the second metacarpal bone. A skiagram was taken of the left hand and wrist and revealed marked osteoporosis of all the bones with two small sequestræ separating from the area of chronic osteo-myelitis of the stump of the second metacarpal bone (see Figure I).

The patient was then referred to the Orthopædic Department, where Dr. D. J. Glissan agreed that massage, active and passive movements and hot air treatments were now indicated to all joints of the left hand as soon as possible. This treatment was begun at once and the patient has made steady progress; there is a moderate degree of movement present (three weeks after the commencement of the active treatment) and the atrophic appearance of the skin is disappearing. The condition of the bones, too, has improved greatly, as can be seen from the skiagram (Figure II), which shows that the stump of the second metacarpal bone has no evidence of periostitis or osteo-myelitis; the sequestræ have been absorbed and the increased density of bones points to regeneration.

[1] Read at a meeting of the New South Wales Branch of the British Medical Association on June 14, 1928.

### Comment.

The case is of interest in showing the advanced grade of osteoporosis which can follow so rapidly upon a mild degree of sepsis and disuse in a hand and the resultant disability. The improvement that followed the institution of active treatment before the septic process had completely settled down, justified this course of action.

### Acknowledgements.

My acknowledgements are due to Dr. T. Maynard Furber who originally saw the patient at the Royal Prince Alfred Hospital; to Dr. Tasman Wilson, who supplied valuable notes about the initial injury and to Dr. Philip Parkinson who reported on the X ray films.

## RENAL DYSTOPIA.

By RICHARD G. S. HARRIS, M.B., Ch.M. (Sydney),
*Honorary Urologist, Lewisham Hospital, Sydney.*

THE following case of dystopia of the left kidney with malrotation, hydronephrosis and stone in the pelvis is of sufficiently rare occurrence to warrant placing it on record.

Mr. N., *ætatis* forty-two years, was admitted to hospital complaining of acute left renal colic of about three days' duration, necessitating hypodermic injections of morphine. On the morning following his admission the colic had subsided, but he complained of some soreness along the course of the left ureter and inability to pass urine, despite the urgent desire. He said he felt there was "something blocking the passage of the urine." The symptoms suggested a descending calculus impacted in the posterior part of the urethra. X ray examination revealed no abnormality. A gum elastic Coudé catheter was introduced into the bladder with some difficulty and tied in.

On cystoscoping the patient's bladder a few days later, after the intravenous injection of indigo-carmine I was unable to find any trace of calculus in the urethra, bladder or ureteral orifices, all of which appeared normal. Urine of a normal blue colour from the right kidney and colourless from the left kidney was collected by the ureteral catheter. The catheter to the left kidney met with some obstruction about ten centimetres (four inches) from the bladder, but after some manipulation passed for another five centimetres (two inches). On pathological examination the urine from both kidneys was sterile and free of pus and blood.

A pyelogram of the left kidney (shown on the special plate) with injection of a 12½% solution of sodium iodide, about twenty cubic centimetres, revealed a most interesting picture. The calyces of the kidney which are incompletely filled, show a marked degree of hydronephrosis. The most interesting feature, however, is the position of the ureter. It will be noted that instead of entering the medial aspect of the kidney, it curves laterally across the kidney and enters the lateral aspect. It will be noted also that the kidney is lying at a lower level and is placed nearer the mid-line than normal.

From this pyelogram and from the cystoscopic findings a preoperative diagnosis of dystopia, hydronephrosis and malrotation was made.

Following this examination, retention of urine was again manifest, but was relieved by catheterization and injection of sixty cubic centimetres (two fluid ounces) of sterile glycerine into the bladder.

Operation was performed one week later by Dr. S. Harry Harris at which I assisted. The usual boomerang lumbar incision was made. On digital exploration it was found that the kidney was lying across the promontory of the sacrum and the left sacro-iliac synchondrosis, with dense fibrous adhesions binding it to the adjacent tissues. The incision was then enlarged by carrying it anteriorly for about five centimetres (two inches) and dividing the oblique abdominal muscles.

With considerable difficulty the kidney was freed from its adhesions and the pathological anatomy was revealed. The kidney was found malrotated, the hilum being directed laterally, the convexity medially. The ventral surface was partially obscured by a grossly dilated pelvis in which a stone was felt and across which an enormous, tortuous, thin-walled, dilated and bifurcating vein ran into the hilum. Running above the vein was an artery. Entering the ventral surface of the lower pole were an artery and vein. An artery and vein were found entering the dorsal surface of the upper pole also and it was only after ligation and division of these vessels and of the ureter that the kidney could be mobilized and completely visualized. Finally, running from the *vena cava* which was now clearly in view, was a dilated vein accompanied by an artery crossing the dorsal surface and entering the dorsal aspect of the hilum of the kidney. Division of these vessels allowed the nephrectomy to be completed.

The semi-diagrammatic drawing (Figure II) shown on the special plate gives a fair representation of the kidney as visualized.

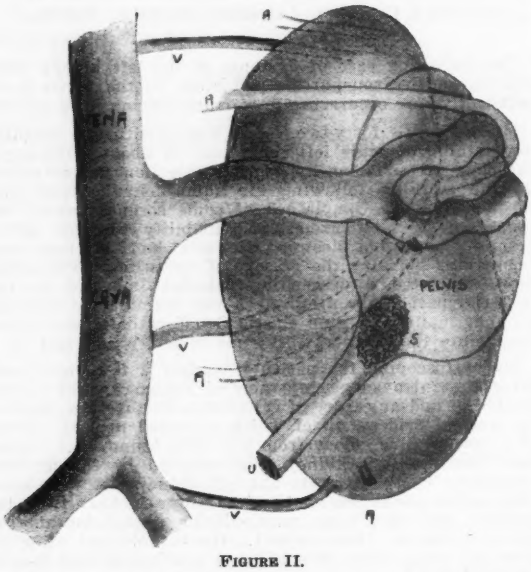


Figure II.

The diagnosis of dystopia from displaced kidney is made by observing the abnormal source of the blood supply to the kidney.

Embryologically, ectopic kidney is explained by failure of the kidney to reach its true position. Actual migration of the kidney is slight and is completed at an early stage. The kidney becomes fixed while the ureter is still quite short—further apparent migration is caused by the rapid growth of the body between the kidney and the vesical end of the ureter. Yet slight though the migration really is, complete or partial failure of it will give rise to a correspondingly marked abnormality in position.

Keyes,[2] quoting Plummer, states:

Whereas a movable kidney is usually on the right side, a misplaced kidney is usually on the left side. The majority of misplaced kidneys are not single kidneys, but the majority of single or of fused kidneys are misplaced.

Young,[1] quoting Naumann, states:

That in 10,177 autopsies there were 20 cases of ectopia—12 on the left side, 5 on the right and 3 bi-lateral. The condition is more common in women than in men.

**References.**

[1] Young: "Practice of Urology."
[2] Keyes: "Urology."

# Reviews.

## INSOMNIA AND DRUG ADDICTION.

Into a monograph of fifty-five pages P. C. Collingwood Fenwick has compressed a great deal about insomnia and drug addiction.[1] Although intended primarily for the general practitioner and medical student, the monograph, because of the simplicity of its language and the delightful absence of highly technical terms, will be readily read by lay people who are interested in such topics.

In Chapter I a list of the various drugs of addiction together with their pharmacological action is given. The author emphasizes the fact that morphine "is not given to produce sleep, but to relieve pain and so allow the patient to sleep," a highly important point which is so often lost sight of by medical practitioners.

In Chapter II the author deals with the causes and effects of insomnia and drug addictions. Physicians who have studied the subject, will agree that "the majority of those who are taking narcotics regularly are not mentally stable," but whilst the assertion that "all drug addicts are curable" may be theoretically true, from the experience of psychiatrists it is a statement that must be accepted *cum grano salis*.

Chapter III is devoted to forms of treatment. The author's treatment of insomnia is more or less orthdox. He is not averse to giving drugs, especially paraldehyde, for insomnia. In the treatment of drug addicts he is opposed to the hyoscine and rapid withdrawal methods. He contends that the gradual withdrawal method is the best method and he claims that its use will give a recovery rate as high as 50%.

A short chapter on after treatment concludes the book. There is no index and no bibliography. The monograph teaches nothing new. It is simply a common sense, readable *résumé* of an admittedly difficult subject and as such can be recommended to practitioners.

## INDUSTRIAL SKIN DISEASES.

To those medical men who act as medical referees or sit on boards under the *Workers' Compensation Act*, the third edition of Dr. Prosser White's book on occupational affections of the skin will be most useful.[2] It is one of the standard works on the subject.

Dr. Prosser White has gone to a great deal of trouble to make this work most thorough. Many of the occupational diseases of the skin mentioned by him are not seen in Australia because we have not the industries concerned. There are one or two conditions which, though mentioned, we would like to know more about, for instance, dermatitis caused by cement and also fungi of various orders. Both these conditions are very common in Australia.

Of the 734 pages 224 are taken up in a very extensive index which is one of the best features of the book. The 500 odd pages of text are divided into twelve chapters under headings of various trades and crafts and irritants. The book is well published.

Although a very complete work on its subject, it is also a difficult book to read on account of the constant use of heavy, thick type which is reminiscent of the habit of certain students who underline passages in their text books until the whole text is underlined. Another annoying feature is the continual quoting of author after author in the text.

We are unable to account for the name given to this work, "The Dermatergoses." It is indeed a new addition to the already much overcrowded and sometimes hideous nomenclature used in the subject of dermatology.

In spite of these irritating features, the work is a much needed one and will be found most useful to anyone dealing with industrial skin diseases.

[1] "Insomnia and Drug Addiction," by P. C. Collingwood Fenwick; 1928. London: H. K. Lewis and Company, Limited. Crown 8vo., pp. 68. Price: 2s. net.

[2] "The Dermatergoses or Occupational Affections of the Skin," Giving Descriptions of the Trade Processes, The Responsible Agents and their Actions, by R. Prosser White, M.D., M.B., C.M. (Edin.), M.R.C.S., (Lond.); 1928. London: H. K. Lewis & Co., Ltd. Demy 8vo., pp. 745. Price: 35s net.

The Medical Journal of Australia

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## The Study of Immunity.

In last week's issue we published three articles in which the authors deal with three separate aspects of immunity. Immunology or the study of immunity, although a relatively new science, has attained such large proportions that the authors were able to discuss only a small part of it and were in consequence unwilling to take a general view of the biological processes associated with infections. It may be of use, notwithstanding the complexity of the subject, to remind ourselves of the phenomena that gave rise to the study. In the first place many of the infective diseases come to an end spontaneously, often after a definite course. In the second place one attack of certain infective diseases confers a considerable degree of protection against a second attack. The third phenomenon is also difficult to explain. Some people can expose themselves to infection without becoming infected. Although these three facts are independent of one another, it is evident that there must be a common factor in their mechanism. The search for an explanation for each of these phenomena led to the discovery of antibody production. This appeared to simplify the study, but it soon became apparent that it did nothing of the sort; it introduced many complicating and puzzling events. The introduction of a pathogenic bacterium into the body leads to the formation of antitoxins, agglutinins, bacteriolysins, opsonins, but it soon became obvious that the appearance of these antibodies in the blood or tissue fluids is not the sole or essential factor in the termination of an infection. Moreover, it was found that under given conditions proteins, whether of bacterial or other origin, can induce hypersensibility instead of insusceptibility to infection. The discovery of anaphylaxis did not render the task less difficult. Further investigation revealed that the processes of immunity are intimately connected with the phenomenon of the incubation period. The doctrines of Ehrlich signalized a new era in the study and even if some of his inferences have long since been proven to be quite untenable, it would have been difficult if not impossible to have obtained any conception of the antigen-antibody reaction had Paul Ehrlich not set up a schematic representation of the underlying bio-chemical characters. When Arrhenius endeavoured to express immunological problems in mathematical terms and challenged the correctness of the interpretations of the physico-chemical findings, it seemed as if further progress had been arrested indefinitely. Continued observation, however, has brought to light a mass of facts, all of which are relevant to the question at issue and some cannot yet be fitted into the mosaic. Twort's researches, later amplified and extended by d'Herelle, promised to provide a relatively simple explanation of the spontaneous termination of certain infections, but as will be gathered from the lucid account by Dr. F. M. Burnet the claims made by d'Herelle have not yet been substantiated and may be coloured by the enthusiasm of an ingenious investigator. The work of Dudley and other epidemiologists must also be taken into account and any explanation offered for the termination of an infection must at the same time be reconciled with the facts relating to the commencement of the infection. Reference has recently been made in these pages to a suggestion that infections may be initiated when the causative organism is present in tissues whose blood vessels are constricted in response to external stimulation of the sympathetic nerve supply. From this it will be gathered that before any hypothesis can be accepted as an explanation of the three facts mentioned above, the greatest care must be exercised in gathering all the known facts and in ascertaining whether these facts can be reconciled with the explanations.

It is assumed that in every infection the infecting agent must be growing in the tissues of the host and it and its products must be powerful enough to overcome the resistance of the host. The mere invasion of the host's tissues by a bacterium either by direct contact or through the intermediation of a vector may be insufficient to produce any illness. It is further assumed that the time occupied between the entrance of the bacterium and the

establishment of free growth of the bacterium corresponds to the incubation period. That the presence of immune bodies in the blood of the host may be the determining factor in the escape from illness of the persons seems to be probable. Further, the resistance of the body may reside in sympathetic control of the surface vessels and it may be the result of complicated metabolic phenomena concerning which so little is known at the present time. It is also assumed that in connexion with cholera, plague, enteric fever, variola, scarlatina, morbilli, syphilis, poliomyelitis, typhus, parotitis, varicella and yellow fever, infections which confer a lasting immunity, a true antitoxin is elaborated and possibly a bacteriolytic antibody as well. It is not suggested that the antibody is sufficient explanation for the spontaneous termination of the infection, when this occurs. Much more work must be done on bacteriophage and on the bio-chemical reactions of infected tissue before an answer to the first question can be given. In regard to those infections against which no lasting immunity is produced, such as pneumonia, dengue fever, diphtheria, tetanus, erysipelas, influenza, tuberculosis and many others, it will be well to postulate that the spontaneous termination when it occurs is caused by antibodies, although this is merely a working hypothesis.

## Current Comment.

### PRIMARY PNUEMOCOCCAL PERITONITIS.

The manner in which primary pneumococcal infection reaches the peritoneum has not been definitely settled. It has been pointed out in these pages on a previous occasion that there are protagonists for the view of hæmatogenous origin, for that of intestinal origin and for the ascent of the infecting organism through the genital passages in the female. McCartney and Fraser have been the chief advocates of the last mentioned view. The evidence which they produced and the arguments which they based on that evidence, were such that many accepted their conclusions without question. Their arguments were not, however, unassailable and in a discussion on the subject we pointed out that it is not beyond the range of possibility that there exists a port of entry, as yet undiscovered, which occasions a general septicæmic infection, and that the presence of organisms in the vaginal secretion and so forth might be a result of this. It was stated to be as reasonable to adopt this assumption as it is to assume on the evidence offered that infection invariably reaches the peritoneal cavity from the vagina. McCartney stated that the literature does not contain the record of a single undoubted primary infection in a boy. In these circumstances every record of an infection regarded as primary and occurring in a male is of importance. D. C. Elkin has reported such a case.[1] He discusses six infections, but the only one which need be considered at the moment, is one which occurred in a negro, aged twenty-nine. The abdomen was opened twice. On the first occasion the appendix was removed, although it was not the seat of definite pathological change; some odourless and slightly flocculent fluid was found in the peritoneum. The fluid was not examined by a pathologist. On the second occasion a large amount of fluid was removed and drainage was established; the fluid contained pneumococci. The whole clinical picture was similar to that seen in females with primary pneumococcal peritonitis. No exploration of the abdomen was made, but it was evident that there was no walling-off of the exudate. The intestine was first reddened and congested and a thin exudate was present; later the fluid became more profuse and contained flakes of fibrin, but did not become purulent until a week after the onset. Elkin states that it cannot be denied that instances of apparent primary infection occur in the male and his general conclusion is that "the primary variety occurs most frequently in young girls, but may occur in boys and men." At the same time he adds that it is possible that there may be present an undiscovered focus elsewhere in the body. If this were so, the condition would not be a primary one. Elkin's report will take its place with those of Montgomery, Leonardo and Annand and Bower who have reported infections in the male held to be primary. Every such report should be considered; eventually the weight of evidence will point in the right direction.

### BLOOD CELLS AND FIBROBLASTS.

As has been pointed out in these pages on several occasions, there is considerable difference of opinion in regard to the origin and the differentiation of the various cells found in the blood. Argument has centred chiefly around the cells of the hyaline series—large and small lymphocytes, large mononuclear cells and the so-called transitional cells. Members of one school of thought hold that these all belong to one series and those of another distinguish sharply between monocytic and clasmatocytic phagocytes. Sabin and her collaborators belong to the latter group. Reference to their work on the monocytes in tuberculosis and to the views of others will be found in The Medical Journal of Australia of December 10, 1927. Since the introduction of tissue culture as a method of study many observers have examined the development of blood cells and some important conclusions have been reached. Lewis has reported the transformation of mononu-

[1] *Archives of Surgery*, February, 1929.

clear cells into macrophages, epithelioid cells and giant cells. Fischer noted the transformation *in vitro* of large mononuclear leucocytes into fibroblasts. Bloom found that lymphocytes of the thoracic duct were transformed into macrophages and Maximow that lymphocytes and monocytes developed *in vitro* into macrophages and fibroblasts. These observations may at first sight appear conclusive, but it is important to remember that, although the conditions obtaining in tissue culture closely resemble those of the body, they are not identical with the latter and caution must be observed in applying laboratory observations to normal or morbid processes in the body. In tissue culture, moreover, a given cell or group of cells may be kept under constant observation. With tissue removed from the bodies of laboratory animals this is not possible and observations must be made from a series of similar lesions.

If these considerations are borne in mind, it will be of interest to discuss a recent communication by G. M. Higgins and B. M. Palmer on the origin of fibroblasts in a hæmatoma produced by experimental methods.[1] They draw attention to the limitations of the method as far as the determination of cell continuity is concerned. At the same time they claim that the conditions prevailing in an experimentally produced hæmatoma are not entirely unlike those attendant on tissue culture. Rabbits were used for the experiments and an aseptic injection of small quantities of autogenous blood was made into the subserous spaces in the walls of the stomach and caecum. The animals were killed at intervals of three, five, seven, eleven, twenty-one, thirty, forty-eight and seventy-two hours. After the tissue had been fixed in a 10% solution of formaldehyde, sections were made and these were stained with hæmatoxylin and eosin. In the earlier hæmatomata the erythrocytes were intact and occasional lymphocytes, monocytes and polymorphonuclear leucocytes were present as in normal blood. In the seven hour hæmatomata slight changes could be noticed. The most pronounced changes were in the nuclei of the large monocytes; they had become more hyperchromatic, their colour resembled somewhat that of the lymphocytes and they had become slightly elongated, though they still possessed their characteristic indented margin. At an early stage the polymorphonuclear leucocytes show signs of destruction. The time and rate of their disappearance, however, is variable. It is concluded that they do not play a part in the subsequent organization or transformation. Higgins and Palmer state that it is evident that the cells which rapidly increase in number, and which possess a variety of shapes ranging from amœboid types with filiform or lobar pseudopodia to forms more distinctly spindle shaped, are not derived from the tissue peripheral to the site of the hæmatoma. This conclusion is formed from the appearance of the sections. These cells are regarded as "probably derivatives of the large monocytes and possibly of the lymphocytes which are normally within the injected blood." There is no evidence that the cells have migrated from the tissues into the blood. The number of cells within the centre of the hæmatoma was even greater than the number in the periphery and the central cells did not show greater differentiation than did those nearer the periphery; Higgins and Palmer think that this condition might ensue if these cells were of extraneous origin. If the cells which become elongated and eventually have all the appearances of fibroblasts have been introduced with the blood injected, they are monocytes; if they have migrated from surrounding tissues, they are clasmatocytic phagocytes, held by Sabin to be derived from endothelium. However probable the truth of a conclusion may be, it is not always wise to accept it because there is no evidence to the contrary. There is one point in the report of Higgins and Palmer which may have a bearing on the question. In hæmatomata of fourteen to twenty-one days' duration the monocytes were more abundant and "had given rise to" amœboid and to spindle shaped cells with somewhat elongated bodies. At thirty hours two rather definite types of cells are present. One is a large oval cell with a single slightly indented nucleus and clear, rather finely granular cytoplasm. This is in the opinion of Higgins and Palmer "unquestionably" the blood monocyte, the clasmatocyte or the phagocytic histiocyte of Ashchoff. The other type of cell is definitely spindle-shaped with a greately compressed nucleus and with a cytoplasmic body greatly attenuated reaching for a considerable distance among the erythrocytes. This cell is regarded as a fibroblast. It is further stated here that as evidence of a peripheral origin is lacking, the two types of cell must be derived from the injected blood and thus be of hæmatogenous origin. Later on these two cell types are still further differentiated. Variations in pigment content are also noted. Some fibroblasts are found to contain pigment and it is presumed that the clasmatocytes became phagocytic before the transformation into fibroblasts took place. It is thought that the large fibroblasts free of pigment are probably transformed monocytes which had not ingested foreign particles and that the smaller fibroblasts may be of lymphocytic origin. In view of the work of others previously mentioned, it is not unlikely that transformation of monocytes and lymphocytes may take place as these authors suggest. It is equally likely, however, that the cells which are found stuffed with pigment, are phagocytic cells of endothelial origin which have entered the affected area from outside. The increase in numbers of the cells in the hæmatoma has to be explained. Higgins and Palmer are evidently satisfied that the increase is due to multiplication of introduced blood cells. It is inconceivable that the tissues will tolerate the presence of a foreign body, even if it be only autogenous blood, without reacting to the stimulus.

[1] *Archives of Pathology*, January, 1929.

# Abstracts from Current Medical Literature.

## PHYSIOLOGY.

### Gastric Acidity.

H. MacLean and W. J. Griffiths (*Journal of Physiology*, December, 1928) have brought forward experimental evidence to show that there is an automatic regulation of the gastric acidity. They consider that their experiments justify the conclusion that secretion of hydrochloric acid by the gastric glands is a self limiting process in that as the gastric acidity or the hydrogen ion concentration increases, it approaches a point at which by virtue of its concentration further secretion is stopped. The presence of a certain concentration of acid inhibits secretion of acid with the result that a neutral fluid containing chloride is secreted which by dilution reduces the acidity of the gastric contents. Inhibition of acid secretion is produced when hydrochloric, sulphuric and acetic acids are introduced into the stomach in concentration greater than the maximum acidity normally found in the stomach. When a fluid is taken into the stomach, this organ by the combined effects of emptying and secretion tends to adjust the concentration of chlorine ions to the normal maximum level (0·11 to 0·12). The peptic activity of the stomach rises parallel with the increase in neutral chloride which occurs along with diminishing acidity.

### Human Skin Temperature.

Under normal conditions of rest about 75% of the heat lost from the body is from the skin chiefly by radiation and 25% is by vaporization of water from the lung and skin. Hence the measurement of skin temperature of itself becomes suggestive in studying the conditions of heat loss. F. G. Benedict and H. S. Parmenter (*American Journal of Physiology*, January, 1929) have studied the effect of muscular activity, exposure to cold and wind movement on the skin temperature in man. The skin temperature was determined by an electrical method from twenty or more spots on the body surface in each case and the subjects of the experiments were twenty-six healthy, normal women. Exposure to severe cold with or without strenuous muscular exercise results in a lowering of the skin temperature chiefly of the extremities, a lowering which cannot be counteracted even by a large production of heat in the strenuous exercise of winter sports. During short periods of severe exercise, such as walking indoors on a treadmill or walking upstairs, the metabolism may be increased more than 500% or 600% and yet the peripheral temperature will simultaneously be lowered temporarily. Even when the skin has been previously cooled by the removal of clothing and there has presumably been some restriction of cutaneous circulation, moderately severe exercise for five minutes may raise the metabolism 500% or 600% without causing an increase in skin temperature. In the exercise experiments the influence of wind movement and of changes in the vaporization of water from the skin were eliminated as far as possible. It is possible that one has to deal here with a vaso-constriction of the peripheral vessels, resulting in a temporary transport of blood from the periphery to the muscles, at least in the initial stages of muscular work.

### Blood Pressure in Hyperpnœa.

Swale Vincent and J. H. Thompson (*Journal of Physiology*, November, 1928) have studied the effect of hyperrespiration on the blood pressure in man. Thirty-seven out of forty-one subjects examined in this series manifested a fall of blood pressure during the period of hyperrespiration. The average depression of blood pressure was equivalent to twenty-five millimetres of mercury. In all instances the falls were rapid in onset and developed maximum depth shortly after cessation of forced breathing. The pulse rate remained remarkably constant at normal during hyperrespiration, but immediately after this period there was a rapid acceleration which occurred with dramatic suddenness and persisted for approximately ten seconds and was followed by a pronounced slowing until the rate became subnormal. Shortly afterwards it returned to normal. The subjects manifesting rises of blood pressure after hyperrespiration had abnormally slow pulse rates. Evidence is given to indicate that the falls in blood pressure are due to mechanical interference with the return of blood to the heart and the lowered carbon dioxide tension of the blood acts as a subsidiary factor.

### The Isolation of Secretin.

J. Mellanby (*Journal of Physiology*, September, 1928) has isolated secretin and he describes its properties. The method of isolation is based on the extraction of secretin from the duodenal membrane by absolute alcohol, the adsorption of the secretin by precipitated bile salts and the precipitation of secretin from aqueous solution by dilute acids. Secretin is a polypeptide containing phosphorus. It is slightly soluble in water, freely soluble in dilute alkali. Secretin in aqueous solution is rapidly destroyed by dilute acid and alkali at 100° C. and by pepsin, trypsin and autolytic enzymes at 38° C. Secretin acts most efficiently when intravenously injected; it produces a slow secretion of pancreatic juice when administered subcutaneously to the unanæsthetized animal; it is not absorbed from the alimentary canal even in the presence of bile. It has no action on the glucose of the blood. Secretin increases the tone of smooth muscle in a concentration of one in 500,000 approximately; but secretin in concentrations in the blood which produce a maximal secretion of pancreatic juice (one in 5,000,000 approximately) does not influence the general plain muscle of the body. Secretin has no action on arterial blood pressure nor on the respiratory system nor the voluntary muscles.

### Muscular Activity.

A. V. Bock, C. Vancaulaert, D. B. Hill, A. Folling and L. M. Hurxthal (*The Journal of Physiology*, October, 1928) have studied in great detail the dynamic changes occurring in man at work. From the standpoint of the body as a machine the results obtained in this study show the great advantage accruing from physical training. The superiority of the athlete lies in his ability to meet the demand for oxygen, enabling him to maintain an internal environment varying within narrow limits only from the resting state. The increased metabolism resulting from muscular exercise is met effectively only in the trained subject, through the coordination of a number of factors, the response in general being about what might be expected in a well integrated system accustomed to such demands. Physical training increases the lung capacity, induces a slow pulse rate, increases the stroke volume of the heart, reduces systemic blood pressure during work and probably greatly increases the capillary diffusion area in the muscles, as it does in the lungs. Coordination of these and other unknown factors by the nervous system results in the provision of an adequate supply of oxygen to meet the needs of the organism up to levels of relatively severe work, with the maintenance of optimum conditions within the body for long periods of time. In the untrained subject these mechanisms may be said to be relatively undeveloped. This statement applies less to the lungs than to the vascular system. Failure to meet oxygen demands in such a subject is reflected within a comparatively short time in the accumulation of lactic acid, diminution of blood bicarbonate, a falling hydrogen ion concentration and a tendency to reach maximum pulse and respiratory rates at levels of work that produce almost no embarrassment for the athlete.

### Cooling Power and Effective Temperature.

J. Argyll Campbell and T. C. Angus (*The Journal of Industrial Hygiene*, December, 1928) in continuation of the study of problems of ventilation by means of Leonard Hill's kata-thermometer have investigated some of the body reactions occurring at various temperatures, namely, about 20°, 25°, 30° and 35° C., in both relatively dry and moisture laden air, still and in motion, using both naked and clothed subjects. They were interested particularly in determining the losses in weight due to the water which has evaporated from the skin and lungs, and also in testing the kata-thermometer against the effective temperature method of Yaglou and his colleagues as an indicator of atmospheric conditions suitable for human beings. Pulse rate, skin temperature, perspiration (insensible and sensible), metabolism and heat loss were studied.

Increase in temperature within the limits specified as a rule caused an increase in pulse rate, skin temperature, perspiration and amount of heat lost by evaporation, while heat loss by conduction, convection, and radiation decreased. Wind opposed these effects in naked subjects only. Clothes increased these effects and antagonized the action of wind. Movement of air and removal of clothes are the obvious remedies for such conditions. Insensible perspiration increased from about twenty to thirty grammes per hour at 20° C. to over one hundred grammes per hour at 35° C.. At this temperature the body gets rid of water not merely for evaporation, but also because wetting the skin aids conduction of heat and because loss of water enables the body to cool down more rapidly, since water has a high specific heat. Moisture in the air, as compared with dry air, has no pronounced effect on perspiration at the higher temperatures studied. At 35° C. metabolism may or may not be increased. Wind 150 metres (500 feet) per minute at 20° C. definitely increased metabolism in naked subjects owing to shivering; at higher temperatures there was no effect of wind on metabolism even in naked resting subjects. Judging from the evidence afforded by pulse rate, water evaporated and cheek temperatures, the dry kata-thermometer cooling power is as good an indicator of physiological reactions to atmospheric conditions as is effective temperature. The kata-thermometer has the advantage of being also an excellent anemometer. For such a comparison of two methods physiological reactions are a better guide than comfort sensations.

## BIOLOGICAL CHEMISTRY.

### Calcium Metabolism in Bone Diseases.

H. K. BARRENSCHEEN AND E. GOLD (*Wiener Medizinische Wochenschrift*, October 20, 1928) have summarized their investigation of the calcium metabolism of the body associated with various diseases of the skeletal system. Estimations of the calcium content of the blood serum and the amount of calcium excreted in the urine during twenty-four hours were made. Only conditions with no opening on the surface of the body were included. The first group consisted of cases of inflammatory disease which were controlled by radiological examination. An increase was noted both in serum calcium and calcium in the urine, but the latter was greater. Similar results were observed with malignant disease, especially with metastatic growths. This was in striking contrast to the results obtained in parasyphilitic bony lesions in which the figures were normal or subnormal. In Paget's disease, while the blood calcium content was high, the output of calcium in the urine was comparatively low. They consider that the test has more than a theoretical interest and that it will prove of value in the differential diagnosis between tabetic lesions and malignant disease.

### Fatness and Sterility.

A. S. PARKES AND J. C. DRUMMOND (*British Journal of Experimental Pathology*, April, 1920) have investigated the relation which exists between extreme fatness in mammals and complete or partial sterility. Sixty-three female rats were put on fat diet and twenty-nine were kept on normal food for mating with dieted males. Some of the dieted females failed to thrive and died, a few failed to respond to the treatment and were killed, fifty rats being left available for further experiment. Twenty were mated with normal males to observe the effect of the diet on fecundity, fertility and sex ratio. Twelve male rats were put on fat diets. One died early and one was dissected after twenty-nine days to ascertain the effect of the diet and ten were mated with normal fertile females. The abdominal fat percentage was used as the index of fatness attained. The detection of all phases of the œstrous cycle was carried out by means of the vaginal smear technique. The animals kept on the fat diets increased their abdominal fat percenfage by more than 100%, but this failed to have any obvious effect on the œstrous cycle. After four months on the fat diet 65% of the females manifested a normal breeding performance; with the males, however, more effect of the diet was found, as the majority of the animals failed to breed with normal females. This is thought to have been due to lethargy caused by the fat condition, as the tests were normal. The authors infer from this result that excessive fatness does not lead to sterility and that fatness is more often a result than a cause of sterility.

### Copper and the Hæmoglobin Content of Rat Blood.

J. S. MCHARGUE, D. J. HEALY AND E. S. HILL (*Journal of Biological Chemistry*, August, 1928) present a preliminary report on the function of copper in the formation of hæmoglobin. Livers of young calves were dried, ground and analysed. Other portions of the livers were ashed and treated so that two solutions were made, one copper-free, the other containing the copper. Two lots of white albino rats were fed on skim milk in the morning and whole milk in the afternoon, distilled water was also given. After the animals had lived on this diet for about four weeks, ten cubic centimetres of the solution without copper were added to the skim milk given to one lot of rats and a like amount of the solution containing copper was added to the skim milk given to the second lot of rats and at the end of the sixth week hæmoglobin estimations were made from both lots of rats. The hæmoglobin readings from the rats receiving the solution containing copper were considerably higher than those from the rats receiving the solution without the copper. The rats that received the copper in their diet, possessed to a noticeable degree a pink ruddiness where the skin is exposed, which was absent on those from which copper was withheld. The blood from the rats from which copper was withheld, was darker in colour than the blood from the rats that received copper, the latter's blood being a brighter red. From these results the authors infer that copper has an important function in the formation of hæmoglobin. Further experiments concerning the effect of copper, manganese, zinc *et cetera* are being carried out.

### Calcium and Phosphorus in Milk.

MARTHA M. KRAMER, ESTHER LATZKE AND MARY MARGARET SHAW (*Journal of Biological Chemistry*, September, 1928) have conducted a series of metabolism experiments with children and adults as subjects to learn whether or not the calcium and phosphorus are equally available in raw, pasteurized, evaporated and dried milks. The results of experiments with five children show that the calcium retention during the periods of consumption of dried milk averaged only 53·71% as much as during the fresh milk period, other factors remaining unchanged. The adult subjects yielded more favourable calcium balance when the fresh milk was used than with the dried milk. Pasteurized milk also gave less favourable calcium balances than did fresh milk. Also the milk from cows kept in a barn for the five months previous to the experiments gave less favourable calcium balances than did the milk from cows in the college dairy. Adults using evaporated milk, however, gave balances as good as when fresh milk was used. Phosphorus balances followed the trend of the calcium balance figures.

### Vitamin D and Blood Chemistry.

R. E. HAVARD AND J. C. HOYLE (*Biochemical Journal*, Volume XXII, Number 3) have investigated the suggestion that the fall of the level of inorganic phosphate in the blood during the winter is due to a slight deficiency of vitamin *D*. Two subjects were given four milligrammes of irradiated ergosterol in the form of "Radiostol" (British Drug Houses) pellets every day for eleven and thirteen days respectively and a third was given eight milligrammes daily for twenty-one days. A fourth subject was kept as a control. The serum calcium and inorganic phosphate of the blood were estimated every day for a week before the feeding began and during the experiment. The blood samples were taken in the mornings and after not less than one hour's rest. No significant changes were found in the inorganic phosphate of the blood or serum calcium. In the second series of experiments two subjects were irradiated for a total period of sixteen days, but no change could be found in the levels of the inorganic phosphate of the blood or serum calcium. These results cast doubt on the conception that the summer rise in the level of the inorganic phosphate of the blood in adults is due to increased incidence of ultra-violet light.

# British Medical Association News.

## SCIENTIFIC.

A meeting of the Victorian Branch of the British Medical Association was held at the Anatomy Department of the University of Melbourne on November 30, 1928. The meeting took the form of a series of demonstrations.

### The Floor of the Female Pelvis.

Professor R. J. A. Berry demonstrated by means of models, diagrams, dissections and lantern slides some points in the muscular mechanics of the female pelvic floor. He pointed out that the names and anatomical descriptions of the perineal and pelvic floor muscles had come down from the ages, when anatomists had had only the naked eye on which to rely. Modern investigations had shown that the descriptions and names of these muscles, still unfortunately in everyday use, were misleading and masked the true physiological and clinical import of these important pelvic elements. Professor Berry's main points were illustrated from the accompanying table showing the elementary enumeration of the principal muscles concerned:

| Topographical Positions and Original Names. | Functions as now Known. |
|---|---|
| A. Anal triangle. | |
| External sphincter of anus | Complex sphincter of anal canal. |
| B. Superficial perineal pouch. | |
| Superficial transverse perineal | Complex sphincter of anal canal. |
| *Bulbo-cavernosus* | Erector apparatus. |
| *Ischio-cavernosus* | Erector apparatus. |
| C. Deep perineal pouch. | |
| *Compressor urethræ* | Simple reinforcing urethral sphincters. |
| Deep transverse perineal | Simple reinforcing urethral sphincters. |
| D. Pelvic floor. | |
| *Levator ani*: *Ilio-coccygeus* | A simple weight-supporting hammock. |
| *Levator ani*: *Pubo-coccygeus* | A complex weight-supporting hammock and sphincter. |
| *a. Pubo-coccygeus* weight | A complex weight-supporting hammock and sphincter. |
| *b.* Sphincter of rectina | A complex weight-supporting hammock and sphincter. |
| *c.* Sphincter of vagina | A complex weight-supporting hammock and sphincter. |
| *Coccygeus.* *Coccygeus* | An evolutionary remnant which completes the pelvic floor. |

From this table it was demonstrated that the several attachments of these muscles—sphincters and weight supporting—are in reality very different from those currently described in text books and are of great clinical and physiological significance.

### Anatomical Aspect of Medical Procedures.

Dr. F. Kingsley Norris demonstrated the anatomical difficulties of some medical procedures.

The technique of longitudinal sinus puncture in infants was indicated by means of a dissection. This route had the advantage of the vein being large, constant in position and fixed by the two layers of enclosing *dura mater*. As long as the posterior angle of the fontanelle could be palpated, the sinus was entered with ease at an approximate depth of half the width of a fold of the scalp. With excessive moulding of the head or gross rickety deformities the normal position might be distorted. In a few cases the parietal bones seemed to meet without forming any palpable angle. These conditions rendered the operation more difficult.

Dr. Norris proceeded to demonstrate the various methods of obtaining cerebro-spinal fluid from the living subject. A brief historical survey of these methods was given. The lumbar region was selected for spinal canal puncture, as below the level of the first lumbar vertebra the spinal cord was represented by a series of filaments, the *cauda equina*, also in this region the interlamina spaces were widest. The effect of posture on these spaces was demonstrated on the skeleton and a sagittally sectioned specimen clearly indicated the anatomical structures to be considered, the ventral and the dorsal plexus of veins within the spinal canal, the fibres of the cauda and the more remote ventral relations of the aorta and the *vena cava* which on rare occasions had been entered in carrying out the operation in children. One member of the audience had seen bowel content withdrawn at lumbar puncture. Any spinal deformity would add to the difficulty of this procedure. The danger of herniation of the medulla into the *foramen magnum* by the withdrawal of excess of fluid, especially in the sitting position, was demonstrated. The *cisterna magna* was demonstrated and the technique of its puncture indicated. The thumb of the left hand was placed on the spine of the axis. The needle was inserted strictly in the mid-line just above this thumb and passed ventrally and cranially along a plane just above the external auditory meatus (indicated by a pointer in the canal) and the gabella. On rare occasions ossification of the posterior occipito-atlantoid ligament had been described. Ventricular puncture was a simple procedure in an infant with an open fontanelle where a needle could be entered at the lateral angle at right angles to the skin. In adults it was found that if a point be selected seven centimetres cranial and one and a half centimetres lateral to the external occipital protuberance, a needle, passed through a drill hole at this site and passed into the brain parallel to the sagittal suture along a plane, the top of the ear, the inner canthus of the eye, would tap the ventricle at its widest part at a depth of about three centimetres. This was demonstrated on a specimen.

In *paracentesis thoracis* one anatomical consideration was the position of the intercostal vessels and nerves which coursed along the grooves in the caudal border of the ribs and which might be exposed to injury if the needle was entered at this site. A dissection was exhibited showing the position of these structures, also the caudal attachments of the pleuræ. The marked dome of the diaphragm and the abdominal contents in relation to the base of the thorax was indicated with the consequent danger to these structures with a basal puncture.

The position of the internal mammary artery was demonstrated as a possible danger in pericardial tapping or intracardiac injection.

The possibility of injury to the bladder, especially in children where this viscus readily became an abdominal organ, with the procedure of intraperitoneal injection was demonstrated. With a mid-line puncture the risk of perforating the deep epigastric artery was avoided.

### The Conducting System of the Heart.

Dr. J. G. Hayden demonstrated the conducting system of the heart and showed dissections of the bundle system in the sheep's and human hearts. Electrocardiograms were shown demonstrating the varieties of bundle lesions. The anatomical relationship between the left branch of the bundle and the right and posterior aortic cusps was shown and an anatomical reason given for the involvement of the left branch in certain cases of endocarditis affecting the aortic valve.

Dissections of the coronary vessels were shown as well as radiographs of hearts in which the coronaries were injected with an opaque medium. Radiographs showed the different area supply of the right and left coronaries in youth and old age and the extensive anastomotic circulation in the latter age period. Two specimens were shown, one of old age atheroma affecting the proximal portion of the left coronary with partial obstruction and a second showing atheroma of the anterior descending branch of the left coronary artery with thrombus formation and a large aneurysm of the anterior portion of the left ventricle. It was pointed out that this was the most common site of thrombosis. In that particular instance pain had not been a feature, but the patient had had two attacks of congestive failure. This agreed with the symptoms in some of Parkinson's and Bedford's series.

### Anatomy of the Lungs.

Dr. Hayden also showed injection radiographs showing the distribution of the branches of the pulmonary artery and of the main bronchi and the anatomical relationships

between these two systems were displayed. Prints and radiographs were shown illustrating the varying types of bronchiectasis displayed by "Lipiodol" injection. Dr. Hayden favoured the intrapharyngeal route for "Lipiodol" injection and considered it was necessary to cocainize the pharynx in only about 20% of cases. In children he preferred the insertion of a small catheter into the larynx under general anæsthesia.

He also showed an example of an atelactatic lower lobe of the left lung in an adult which had been demonstrated by a small pneumothorax as suggested by Dr. Singer, of St. Louis. This lobe appeared as a triangular shadow in the presence of a small pneumothorax, but had not been visible in films taken in the routine manner. The patient had been treated by artificial pneumothorax and had lost all her symptoms.

He also showed films demonstrating that in cases of pulmonary abscess with secondary empyema the position of the abscess could be ascertained by removing a little fluid and inserting air and taking the film with the patient lying on the good side. By this method the fluid fell into the mediastinal hollow and cleared the lung. By tilting the patient varying parts of the lung could be cleared. As a rule these patients were too ill to have films taken in the ordinary upright position and if films were taken in the supine position, a small amount of fluid would obscure the whole lung field.

### The Upper Part of the Abdomen.

Mr. A. E. Coates demonstrated the anatomy of the peritoneal spaces of the upper part of the abdomen. The depth of the recto-vesical pouch and the necessity for drainage of that cavity in inflammatory conditions were emphasized. A study of the abdominal wall and the direction in which effusions would gravitate, showed the fallacy of drainage of the iliac fossa. Morrison's pouch and the right anterior peritoneal subphrenic space were demonstrated, stress being laid on the posterior position of the right triangular ligament which is attached to the liver far back near the angles of the ribs.

The anterior space then could be effectively drained from behind in the posterior axillary line by a transpleural approach after resection of the ninth or tenth rib. The omental bursa and the surgical approach to the pancreas were demonstrated and the arteries of the upper part of the abdomen traced to their ultimate distribution.

### Brain and Spinal Cord.

Mr. W. A. Hailes first demonstrated a head in which the scalp had been reflected and all the bones removed as windows, leaving the sutures and a small area of bone around each suture. The membrane had then been removed and the sulci and convolutions of the brain exposed. The specimen clearly showed the relationships of the important fissures of the brain to the suture lines on the skull, this being especially valuable in the case of the relationship of the central sulcus to the coronal suture and the sylvian fissure to the parieto-temporal suture and the pterion.

Mr. Hailes then demonstrated a head in which the frontal lobe and roof of the orbit had been removed on one side to show all the relationships of the pituitary fossa from above and to illustrate the anatomy of the Frazier approach to the pituitary tumour and the anatomy of the optic nerve and chiasma.

A third head specimen had been dissected to show the anatomy of the posterior fossa of the skull as seen in Cushing's approach. On one side portion of the cerebellum had been removed and clearly showed the posterior aspect of the petrous portion of the temporal bone with the internal auditory meatus and the fifth, seventh and eighth and ninth, tenth and eleventh cranial nerves and their relationship to the cerebello-pontile angle. In the same specimen was displayed the anatomical relationship of the *cisterna magna* with special reference to the interval between the neural arch of the atlas vertebra and the posterior aspect of the medulla, a point of great importance in *cisterna magna* puncture.

Mr. Hailes's fourth specimen was a dissection of the *cauda equina* designed to show the anatomy of the centrally placed *cauda equina* tumour and of the laterally placed tumour; also the anatomy of the mixed cord and *cauda equina* injury. In this specimen also could be seen the relationship of the first lumbar nerve to the Y-shaped bifurcation of the dentate ligament and apart from that anatomical landmark there was demonstrated the extreme difficulty of identifying the nerve roots of the *cauda equina* through any limited exposure such as was seen at operation.

## MEDICO-POLITICAL.

### A Correction.

The Council of the New South Wales Branch of the British Medical Association has asked that it may be announced for the information of members of the Branch and in particular of the members residing in the localities indicated that there is no authority whatever for the statement published in a recent issue of a Sydney morning daily that the British Medical Association had given its approval to a proposal that Dr. — and Dr. — should visit districts in the far west and carry out inspections of crippled persons. At the same time the Council wishes to make it clear that the practitioners referred to were not responsible for the statement.

## NOMINATIONS AND ELECTIONS.

The undermentioned have been nominated for election as members of the New South Wales Branch of the British Medical Association:

Graham, Colin Spencer, M.B., Ch.M., 1926 (Univ. Sydney), Lane Cove Road, Turramurra.

Boag, James Fraser, M.B., Ch.M., 1925 (Univ. Sydney), 92, Birkeley Road, Manly.

The undermentioned has been elected a member of the Victorian Branch of the British Medical Association:

Darbyshire, Joan, M.B., B.S., 1928 (Univ. Melbourne), Melbourne Hospital.

# Correspondence.

## MEDICAL HISTORY OF THE WAR.

Sir: I should be grateful of your cooperation, through the medium of your correspondence column, in obtaining illustrations of certain aspects of the work of the Australian Army Medical Corps for Volume I of the Australian Medical History of the war. Though through the Australian War Memorial and personal sources many excellent photographs are available, some aspects of the work and experiences of the Corps are imperfectly covered. The subjoined list indicates subjects on which among others photographs (or even better, black and white drawings or sketches) would be gratefully received by me (addressed to the Defence Department, Melbourne) and carefully returned. To be of use response should be prompt.

Yours, etc.,

A. G. Butler,
Medical Historian.

*Subjects.*

In general pictures are required illustrating:

(*a*) The training and development of the Australian Army Medical Corps (including associated services) in Australia and abroad during the Great War and also during the early history of the service.

(*b*) Sites or positions occupied by medical units in the field, lines of communication and at the

bases (including Australia and England), with the purpose in view of showing the conditions under which they worked.

(*c*) Illustrating the work of medical units, first aid and immediate treatment, collection, clearance, reception, treatment and disposal of sick and wounded.

(*d*) "Sanitation"—in particular sanitary arrangements in the field and work of sanitary units and establishments.

In particular pictures illustrating the work of medical units and personnel during actual operations are desired.

In the case of pictures or collections which by reason of size would involve any special difficulty in transmission, to avoid needless trouble by reason of duplication a note might be sent stating the subjects illustrated and nature of the pictures; further action to depend on reply received.

## FORCED DRAINAGE OF THE CENTRAL NERVOUS SYSTEM.

Sir: It gave me great pleasure to read your comments on "Forced Drainage of the Central Nervous System" on page 536 of the issue of October 27, 1928, of your journal.

In your discussion, however, it became clear that two points were causing you misgivings which could be readily disposed of by a word of further explanation.

1. It is evident that the reviewer was puzzled to know how intracranial pressures could be measured while free escape of cerebro-spinal fluid was permitted. In reply to this, it should be noted that in the article under review hydration alone was studied, the pressure changes having been made the object of a separate investigation at an earlier date. Reference is made to this earlier study in the present article, however, and a cut from the earlier study is reproduced to show the method employed. (*Confer*, *Archives of Neurology and Psychiatry*, September, 1926, Volume XVI, pages 319 to 328.)

2. The reviewer also was under the impression that data on venous pressure had been included in order to give an indirect index of the changes in intracranial pressure. This had not been the intention; because although an increase in venous pressure will always raise intracranial pressure, it has been shown that intracranial pressure can rise without any corresponding change in venous pressure. The object of including the data on venous pressure was to demonstrate that even massive volumes of these hypotonic solutions could be injected without overburdening the heart, provided the injection was slowly given. The fact that under these conditions the venous pressure did not rise is a most sensitive indicator that the circulation was not unduly strained.

Yours, etc.,

Lawrence S. Kubie, M.D.

17, Langham Street, London, W.1.
January 27, 1929.

## MYXŒDEMA.

Sir: A female, *ætatis* sixty-two years, consulted me six months ago, stating that for the last eight years she had suffered from "huskiness" and periodical loss of voice.

She had been advised to take a lengthy sea voyage as her complaint was due to nerve exhaustion. This trip (to England) she took, but her health still was in the same state. She then, as her home was at Bondi, left the district and came to live at Roseville, where the climate might be more suitable. She derived no benefit, though she thought the laryngitis was better, but the hoarseness still persisted.

On examination I found the skin to be much roughened over the elbows and back of the chest and her gait was of the "waddling" type. In addition, the expression of the fact was mask-like. I could detect no circulatory or faucial or any constitutional defect.

I put her on thyreoid gland tablets (one grain) thrice daily and within three weeks the huskiness and laryngeal attacks quite disappeared. She noticed too that she seemed different and was able to walk better. It is now six months since prescribing and she keeps remarkably well, but takes one grain of thyreoid every day.

I take this to be a case of myxœdematous infiltration of the larynx which was cured by the exhibition of thyreoid gland.

Yours, etc.,

A. M. Watkins.

74, Bancroft Avenue,
Roseville, New South Wales,
February 16, 1929.

## SNAKE BITE.

Sir: I was much interested in Dr. Hamilton Fairley's article "Symposium of Snake Bite" in your issue of March 9 last.

I would like to call attention to a deadly venomous snake *Oxyuranus maclennani* (Kinghorn) discovered by Mr. William McLennan on March 31, 1922, on Cape York Peninsula while on an ornithological expedition on behalf of the late H. L. White, of "Belltrees," Scone, New South Wales, which was reported in *The Emu*, Volume XXII, Part 2 (published in October, 1922, by the latter and mentioned again in *The Emu*, Volume XXIII, Part 2, October, 1923), by Mr. Roy Kinghorn, Zoologist to the Australian Museum. Two specimens—a skull and skin of one and a skull of another—were sent to the Australian Museum. The length of both snakes was nine feet and the poison fangs were stated to be "so large they would easily penetrate through one's finger if bitten," to do which they would have to be at least fifteen millimetres (three-fifths of an inch) in length. Compare this with that of the death adder (six millimetres) and one realizes what a dangerous snake it is.

From the length of the fangs and the arrangement of the cranial bones, this species approached more closely to the family *Viperidæ* than any of our snakes. It would be interesting to study this snake alive and compare the potency of the venom, the amount injected at a bite and its lethality with our well known deadly snakes and with the African black mamba and puff adder.

Yours, etc.,

E. C. Chisholm, M.B., Ch.M.

Comboyne,
March 11, 1929.

# Obituary.

## ROBERT SAMUEL TROTTER.

It is with regret that we have to announce the death of Robert Samuel Trotter which occurred on March 10, 1929, in Sydney. He was born in Perth, Scotland, in 1870. His father, Robert Bruce Trotter, was a medical practitioner in that city. The son studied medicine in Aberdeen and took the degrees of bachelor of medicine and master of surgery in 1898. Later he obtained the diploma of public health and degree of doctor of hygiene at the University of Durham. In 1903 he obtained his doctor of medicine. After graduation he succeeded his uncle in practice at Bedlington in Northumberland, where he became medical officer of health. After a time he was elected a member of the Northumberland County Council. He remained in practice until 1914 when he was appointed medical officer in Fiji. In 1916 he became chief medical officer of Raratonga, Cook Islands. He worked in the islands of the Pacific until 1925 when he secured an appointment as medical officer of health at Broken Hill, New South Wales. He resigned after six months on account of ill health. He made some valuable contributions to the literature on miners' phthisis and the article on

this subject in the "Encyclopædia Medica" was written by him.

He is survived by his widow and a son and a daughter. The sympathy of a large circle of friends in Scotland, the north of England and the Pacific islands is extended to them.

## Research.

### THE KATHERINE BISHOP HARMAN PRIZE.

THE Council of the British Medical Association announces that it is prepared to consider an award of the Katherine Bishop Harman Prize in the year 1930. The value of the prize is £80. The purpose of the prize is the encouragement of study and research directed to the diminution and avoidance of the risks to health and life that are apt to arise in pregnancy and child bearing. Competitors are free to select the work they wish to present, provided that it falls within the scope of the definition. Any medical practitioner registered in the British Empire is eligible to compete for the prize. Should the Council decide that no essay submitted is of sufficient merit, the prize will not be awarded in 1930, but will be offered in the year following that in which the decision is made. In this event the money value of the prize will be determined by the Council.

The essay must be typewritten or printed in the English language. It must be distinguished by a motto and must be accompanied by a sealed envelope marked with the same motto on the outside and enclosing the author's name and address. The essays must be forwarded to the Medical Secretary, British Medical Association House, Tavistock Square, London, W.C.1. They must be in his hands not later than December 31, 1929. Inquiries relative to the prize should be addressed to the Medical Secretary.

## Proceedings of the Australian Medical Boards.

### NEW SOUTH WALES.

THE undermentioned have been registered under the provisions of *The Medical Act*, 1912 *and* 1915, of New South Wales as duly qualified medical practitioners:

Lynch, Vincent Thomas Joseph, L.L.M.R.C.P. (Ireland), 1927; L.L.M.R.C.S. (Ireland), 1927; 789, George Street, Sydney.

Goode, Arthur, M.B., B.S., 1894 (Univ. Adelaide), c.o. Dr. Simmons, Forest Road, Bexley.

Jones, John Christopher, M.B., Ch.B., 1913 (Univ. Birmingham); M.R.C.S. (England), 1914, L.R.C.P. (London), 1914.

Pardey, Geoffrey James, M.B., B.S., 1928 (Univ. Melbourne), Prince of Wales Hospital, Randwick.

Reeves, Rupert Kirk, M.B., B.S., 1926 (Univ. Adelaide), 60, Lang Terrace, Centennial Park.

Rock, William, L.R.C.P. (Edinburgh), 1894, L.R.C.S. (Edinburgh), 1894, L.F.P.S. (Glasgow), 1894.

Rogers, William, M.B., Ch.B., 1900, M.D., 1911 (Univ. Edinburgh), Araluen.

Phillips, Lewis Leon, M.B., B.S., 1927 (Univ. Melbourne), Wagga.

Sullivan, Arthur Bernard, M.R.C.S. (England), 1928; L.R.C.P. (London), 1928; Saint Vincent's Hospital, Sydney.

For additional registration:

Maddox, John Kempton, M.R.C.P. (London), 1928.

### QUEENSLAND.

The undermentioned have been registered under the provisions of *The Medical Act of* 1925, of Queensland, as duly qualified medical practitioners:

Gargiulo, Francesco, M.D., 1910 (Univ. Naples), Einasleigh.

Ross, Keith Chisholm, M.B., B.S., 1923, Ch.M., 1928 (Univ. Melbourne), Brisbane.

McColl, Bernard Howard, M.B., B.S., 1928 (Univ. Melbourne), Brisbane.

Unwin, Maurice Leslie, M.B., Ch.M., 1925 (Univ. Sydney), Many Peaks.

Parker, William Patrick Hugh, L.R.C.P. *et* S. (Ireland), 1910, Brisbane.

Crowe, Patrick Francis Vincent, Lic. Lic. Midwif., R.C.P. & S. (Ireland), 1912; Brisbane.

Haynes, Raymond James, M.B., 1915 (Univ. Sydney), Brisbane.

Walker, James Duncan, M.B., Ch.M., 1891 (Univ. Aberdeen), Tambo.

Chalmers, Alexander William, M.B., 1922 (Univ. Sydney), Goondiwindi.

Restoration to the Register:

Cunningham, Anthony Benedict, M.B., Ch.M., 1921 (Univ. Sydney), Gympie.

### VICTORIA.

THE undermentioned have been registered under the provisions of Part I of *The Medical Act*, 1915, of Victoria, as duly qualified medical practitioners:

Killmier, Ernest Raymond, M.B., B.S., 1923 (Univ. Melbourne), 432, St. George's Road, Thornbury.

Kneebone, John Le Messurier, M.S., 1921 (Univ. Adelaide), F.R.C.S. (England), 1921, Hamilton.

Schafer, David Paul Hannaford, M.R.C.S. (England), L.R.C.P. (London), 1927, c.o. National Bank, Collins Street, Melbourne.

Schafer, Noel Theodore Hannaford, M.R.C.S. (England), L.R.C.P. (London), 1927, c.o. National Bank, Collins Street, Melbourne.

Additional diploma registered:

Blanch, Mona Margaret, M.D. (Melbourne), 1926, M.R.C.P. (London), 1928.

### TASMANIA.

THE undermentioned have been registered under the provisions of *The Medical Act*, 1918, of Tasmania, as duly qualified medical practitioners:

Robertson, Fanny Croaker, M.B., Ch.M., 1926 (Univ. Sydney), St. Helens.

Short, Frederick, M.B., 1916 (Univ. Sydney), Ch.M., 1921 (Univ. Sydney), Hobart.

### MEMORIAL TO THE LATE HARRY JOHN CLAYTON.

THE following additional subscriptions have been received by the Honorary Treasurers of the Harry John Clayton Memorial Fund:

| | £ | s. | d. |
|---|---|---|---|
| Previously acknowledged .. .. .. | 464 | 13 | 9 |
| Dr. A. L. Dawson .. .. .. .. .. | 15 | 15 | 0 |
| Miss M. D. Clayton .. .. .. .. .. | 10 | 0 | 0 |
| Professor A. E. Mills .. .. .. .. | 5 | 5 | 0 |
| H. S. Reid .. .. .. .. .. .. .. | 5 | 5 | 0 |
| Dr. W. B. Thompson .. .. .. .. | 5 | 0 | 0 |
| Dr. R. A. Noble .. .. .. .. .. | 3 | 3 | 0 |
| T. A. J. Playfair .. .. .. .. .. | 3 | 3 | 0 |
| Dr. C. H. Shearman .. .. .. .. | 2 | 2 | 0 |
| Dr. W. S. Dawson .. .. .. .. .. | 1 | 1 | 0 |
| Dr. Karen Helms .. .. .. .. .. | 1 | 1 | 0 |
| E. R. Mitchell .. .. .. .. .. .. | 1 | 1 | 0 |
| Dr. C. W. W. Murray .. .. .. .. | 1 | 1 | 0 |
| Dr. E. A. Tivey .. .. .. .. .. | 1 | 1 | 0 |
| Total to date .. .. .. .. .. | £519 | 11 | 9 |

## Books Received.

SURGERY IN THE TROPICS, by Sir Frank Powell Connor, D.S.O., F.R.C.S., D.T.M. & H.; 1929. London: J. and A. Churchill. Post 8vo., pp. 301, with illustrations. Price: 12s. 6d. net.

THE PRACTICAL MEDICINE SERIES COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY: Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; The Eye, Ear, Nose and Throat; 1928. Chicago: The Year Book Publishers. Crown 8vo., pp. 540, with illustrations.

THE PRACTICAL MEDICINE SERIES COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY: Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; General Surgery; 1928. Chicago: The Year Book Publishers. Crown 8vo., pp. 800, with illustrations.

THE PRACTICAL MEDICINE SERIES COMPRISING EIGHT VOLUMES ON THE YEAR'S PROGRESS IN MEDICINE AND SURGERY: Under the General Editorial Charge of Charles L. Mix, A.M., M.D.; Pediatrics; 1928. Chicago: The Year Book Publishers. Crown 8vo., pp. 441, with illustrations.

TEXT-BOOK OF PATHOLOGY, by Robert Muir, M.A., M.D., Sc.D., LL.D., F.R.S.; Second Edition; 1929. London: Edward Arnold and Company. Royal 8vo., pp. 890, with illustrations. Price: 35s. net.

SURGICAL PATHOLOGY, by Cecil P. G. Wakeley, F.R.C.S. (England), F.R.S. (Edinburgh), and St. J. D. Buxton, M.B., B.S. (London), F.R.C.S. (England); 1929. Bristol: John Wright and Sons, Limited. Royal 8vo., pp. 930, with illustrations. Price: 45s. net.

A MANUAL OF HELMINTHOLOGY: MEDICAL AND VETERINARY, by H. A. Baylis, M.A., D.Sc.; 1929. London: Baillière, Tindall and Cox. Royal 8vo., pp. 314, with illustrations. Price: 30s. net.

PRESCRIBING OCCUPATIONAL THERAPY, by William Rush Dunton, Jr.; 1928. London: Baillière, Tindall and Cox. Crown 8vo., pp. 149. Price: 10s. net.

COMMON COLDS: CAUSES AND PREVENTIVE MEASURES, by Leonard Hill, M.B., F.R.S., Hon. A.R.I.B.A., and Mark Clement; 1929. London: William Heinemann (Medical Books) Limited. Demy 8vo., pp. 134. Price: 7s. 6d. net.

TEXTBOOK OF CLINICAL NEUROLOGY FOR STUDENTS AND PRACTITIONERS, by M. Neustaedter, M.D., Ph.D., with an introduction by Edward D. Fisher, M.D.; 1929. Philadelphia: F. A. Davis Company. Royal 8vo., pp. 622, with illustrations. Price: $6.00 net.

## Diary for the Month.

April 9.—Tasmanian Branch, B.M.A.: Branch.
April 9.—New South Wales Branch, B.M.A.: Ethics Committee.
April 10.—Federal Committee of the B.M.A. in Australia.
April 10.—Central Northern Medical Association, New South Wales.
April 11.—Victorian Branch, B.M.A.: Council.
April 11.—New South Wales Branch, B.M.A.: Clinical Meeting.
April 12.—Queensland Branch, B.M.A.: Council.
April 16.—Tasmanian Branch, B.M.A.: Council.
April 16.—New South Wales Branch, B.M.A.: Organization and Science Committee.
April 16.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
April 17.—Western Australian Branch, B.M.A.: Branch.
April 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
April 23.—Illawarra Suburbs Medical Association, New South Wales.
April 24.—Victorian Branch, B.M.A.: Council.
April 25.—South Australian Branch, B.M.A.: Branch.

## Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx, xxi.

Launceston Public Hospital: Junior Resident Medical Officer.

Mental Diseases Hospital, New Norfolk, Tasmania: Assistant Medical Officer.

Newcastle Hospital: Resident Radiologist.

New South Wales Branch of the British Medical Association: Medical Secretary.

The Children's Hospital, Incorporated, Perth, Western Australia: Junior Resident Medical Officers.

The University of Melbourne: Chair of Obstetrics.

Victorian Eye and Ear Hospital: Resident Surgeons (three vacancies).

## Medical Appointments: Important Notice.

Medical practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| New South Wales: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District United Friendly Societies' Dispensary.<br>Balmain United Friendly Societies' Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham United Friendly Societies' Dispensary.<br>Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney.<br>North Sydney Friendly Societies' Dispensary Limited.<br>People's Prudential Assurance Company, Limited.<br>Phœnix Mutual Provident Society. |
| Victorian: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| Queensland: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital.<br>Boonah District Hospital. |
| South Australian: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| Western Australian: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| New Zealand (Wellington Division): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

Medical practitioners are requested not to apply for appointments to position at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, New South Wales.

## Editorial Notices.

Manuscripts forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to The Medical Journal of Australia alone, unless the contrary be stated.

All communications should be addressed to "The Editor," The Medical Journal of Australia, The Printing House, Seamer Street, Glebe, Sydney. (Telephones: MW 2651-2.)

Subscription Rates.—Medical students and others not receiving The Medical Journal of Australia in virtue of membership of the Branches of the British Medical Association in the Commonwealth can become subscribers to the journal by applying to the Manager or through the usual agents and booksellers. Subscriptions can commence at the beginning of any quarter and are renewable on December 31. The rates are £2 for Australia and £2 5s. abroad *per annum* payable in advance.